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Clustering of Atherogenic Behaviors in Coffee Drinkers

Elizabeth M. Puccio, MD, Janice B. McPhillips, MS, Elizabeth Barrett-Connor, MD,
and Theodore G. Ganiats, MD

Abstract: We studied the clustering of coffee consumption and selected atherogenic behaviors in older adults living in a southern California community. Men were somewhat more likely to drink caffeinated coffee while women were more likely to drink decaffeinated coffee. In men, but not women, caffeinated coffee drinking decreased with age and decaffeinated coffee drinking increased. Caffeinated coffee drinkers drank more alcohol, consumed more dietary saturated fats and cholesterol, were more likely to be current smokers and less likely to be current exercisers than were non-coffee drinkers. Smoking and exercise also showed a dose-response relationship to the amount of caffeinated coffee consumed. Risk factor levels among drinkers of decaffeinated coffee were more like those of caffeinated coffee than non-drinkers. These data illustrate the clustering of atherogenic behaviors with coffee drinking and highlight their potential importance in interpreting the growing body of literature about coffee and health. (*Am J Public Health* 1990; 80:1310–1313).

Introduction

Whether coffee drinking is a risk factor for coronary heart disease continues to be controversial. Some epidemiologic evidence has linked heavy coffee consumption to elevations in total serum cholesterol or low density lipoprotein cholesterol,[1–3] suggesting a mechanism for the association of coffee and coronary heart disease. Several investigators have reported an increased risk of coronary heart disease morbidity and mortality with heavy coffee intake,[4–8] but others have questioned whether this association is independent of cigarette smoking.[9–12] Only two previous studies[13,14] have examined coffee use in relation to multiple behaviors other than smoking, that could themselves be associated with hyperlipidemia and/or coronary heart disease. In this paper, the coffee drinking habits of a southern California population-based cohort of older men and women were examined in relation to multiple putative heart disease risk factors to determine the extent to which coffee drinking is associated with other atherogenic behaviors.

Methods

Participants were a part of the Rancho Bernardo Heart and Chronic Disease Study, a population-based study designed to examine lifestyle and chronic disease patterns in older adults. All surviving members of the cohort who were between 40 and 84 years of age when first seen in 1972-74 were invited to participate in 1984-87, and 80 percent did so. Height and weight were measured with participants in light clothing without shoes; obesity was estimated using body mass index (kg/m^2). Data on current exercise habits, current and past smoking history, current alcohol consumption, hours of nightly sleep, and use of non-contraceptive estrogens (women only) were determined by a standardized interview administered by trained personnel. Coffee intake and diet data were ascertained as part of a self-administered food frequency questionnaire. Participants were asked to estimate their average consumption of decaffeinated and regular coffee (in cups per day) and their usual intake (as an adult over 45 years of age) of 128 food items.

A crude index of dietary saturated fat and cholesterol intake was made based upon reported consumption of foods selected by the American Heart Association as food to limit in a fat-controlled, low-cholesterol diet.[15] Six responses for frequency of intake ranging from "never" to "daily" were transformed to ranks from "1" to "6" with "1" corresponding to "never" and "6" corresponding to "daily." The cholesterol and saturated fat index was created by computing an integer sum of the participants' coded estimates of consumption for the following 12 foods or food categories: beef, ham, hot dogs, eggs, milk, organ meats, cream, cheese, ice cream, butter, lard, dessert cakes.

Participants were grouped into four coffee drinking categories: those who drank no coffee, drinkers of caffeinated coffee only, drinkers of decaffeinated coffee only, or those who drank both caffeinated and decaffeinated coffee. Participants were also grouped according to whether or not they reported exercising at least three times a week, whether they currently smoked cigarettes, quit smoking, or never smoked, and for female participants, whether or not they reported having taken any oral estrogen within the past two weeks. The total number of bottles or cans of beer, glasses of wine, highballs, cocktails, mixed drinks, liqueurs or other alcoholic drinks consumed was used as an index of alcohol consumption; daily alcohol intake estimated from the past week or from usual intake from the diet questionnaire were nearly identical and only the former is shown. Reported hours of usual nightly sleep were estimated to the nearest hour.

Results are presented for 2,304 men and women ages 50-89. One-way analysis of covariance was used to compare mean age-adjusted risk factor levels among those who drank caffeinated coffee only, decaffeinated coffee only, or both caffeinated and decaffeinated coffee to those who drank no coffee. In addition, mean risk factors for caffeinated coffee drinkers were compared to decaffeinated coffee drinkers. Age-adjusted rates of categorical risk factors were computed by the direct method and differences in age-adjusted rates were tested using the Mantel-Haenszel summary chi-square with one degree of freedom. No adjustments were made to account for the effects of multiple comparisons.

Coffee consumption in those who drank caffeinated coffee only (464 men, 514 women) was divided into three categories as follows: one to two cups per day, three to four cups per day, and

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five or more cups per day. Trends in age-adjusted behavioral risk factor with increasing coffee consumption were tested by analysis of covariance for continuous variables and by the Mantel extension test[16] for categorical variables. Because there were very few heavy drinkers of decaffeinated coffee only, analysis of trends could not be performed.

Results

As shown in Table 1, the 1,028 men and 1,276 women ages 50-89 years were similar with regard to the proportion who drank no coffee (11 percent) or caffeinated and decaffeinated coffee combined (28 percent). Men were somewhat more likely to drink caffeinated coffee only and women to drink decaffeinated coffee only, although both sex differences were small. The proportion of men who drank only decaffeinated coffee increased with age (p for trend <0.001) while the proportion who drank only caffeinated decreased (p for trend = 0.002); similar trends were not observed in women.

Mean age and sex-adjusted behaviorial risk factors for coronary heart disease (CHD) by coffee consumption category are summarized in Table 2. In both men and women, caffeinated coffee drinkers were significantly more likely to be current cigarette smokers and to consume more alcohol and saturated fats and cholesterol than non-coffee drinkers. The percent of women who exercised at least three times per week was lower in all types of coffee drinkers as compared to non-coffee drinkers; the reverse was true for men (data not shown). Neither difference was statistically significant. BMI and hours of nightly sleep did not differ significantly in coffee drinkers as compared to non-drinkers in either sex.

Age-adjusted levels of many risk factors among decaffeinated coffee drinkers were more similar to those who drank caffeinated coffee than to those who drank no coffee (Table 2). However, dietary consumption of saturated fats and cholesterol was significantly higher in caffeinated coffee drinkers as compared to decaffeinated coffee drinkers in both men and women. Women who drank caffeinated coffee had a significantly lower use of replacement estrogen than decaffeinated coffee drinkers (data not shown). Drinkers of caffeinated coffee consumed more alcohol and were more likely to be current cigarette smokers than those who drank decaffeinated coffee.

Among those who drank caffeinated coffee only, the proportion of men and women who currently smoke cigarettes increased with increasing caffeinated coffee consumption (p for trend in age-adjusted rates <0.01 for men, <0.001 for women) (Table 3). The frequency of current exercise decreased with increasing coffee consumption in both men and women although the trend was significant only among men. There were no other clear associations between the amount of coffee consumed and alcohol, dietary cholesterol, hours of nightly sleep, or post-menopausal estrogen use (among women only). The range of decaffeinated coffee drinking (one to two cups) was too narrow to be considered in a similar analysis.

Discussion

Coffee drinking may be a marker for an atherogenic diet, cigarette smoking, or sedentary lifestyle, rather than an etiologic factor in heart disease outcomes. For example, Hennekens, *et al.*,[10] reported a positive association between coffee drinking and mortality from coronary heart disease, independent of cigarette smoking, that disappeared with adjustment for daily alcohol consumption, level of physical activity, and use of coffee additives.

In the present study, there was an increased occurrence of three habits in caffeinated coffee drinkers: use of cigarettes, excess consumption of alcohol, and an atherogenic diet. There was a dose-response relationship in the extent to which some of these behaviors were practiced among drinkers of caffeinated coffee.

These results support the findings of Jacobsen and Thelle,[13] Haffner, *et al.*,[14] and others[17,*] who found that coffee drinkers consume a more atherogenic diet than their non-coffee drinking counterparts. Carmody, *et al.*,[18] also reported that smokers and ex-smokers were more likely than non-smokers to drink greater amounts of alcohol and coffee; most individuals who reported drinking more alcohol also reported drinking more coffee, regardless of smoking habit. In a study of an industrial population, Phillips, *et al.*,[19] noted that smokers drank an average of 1.5 to 2 more cups of coffee per day and drank more alcohol weekly than non-smokers.

Others have reported that coffee drinkers have a decreased body mass relative to non-coffee drinkers.[20] In this cohort, body mass index did not differ by coffee intake, but those who drank five or more cups of caffeinated coffee per day exercised significantly less than those who drank less. Leon, *et al.*,[21] found that the use of caffeine-containing

TABLE 1—Distribution of Coffee Consumption According to Age and Sex, Rancho Bernardo, 1984–87

		No Coffee		Decaffeinated Only		Caffeinated Only		Both	
Sex, Age (years)	N	n	%	n	%	n	%	n	%
Men									
50–59	166	27	16.3	16	9.6	83	50.0	40	24.1
60–69	254	30	11.8	31	12.2	129	50.8	64	25.2
70–79	404	30	7.4	63	15.6	175	43.3	136	33.7
80–69	204	24	11.8	56	27.4	77	37.7	47	23.0
Total	1028	111	10.8	166	16.1	464	45.1	287	27.9
Women									
50–59	209	29	13.9	42	20.1	86	41.1	52	24.9
60–69	339	34	10.0	76	22.4	134	39.5	95	28.0
70–79	539	52	9.6	114	21.2	215	39.9	158	29.3
80–89	189	23	12.2	41	21.7	80	42.3	45	23.8
Total	1276	138	10.8	273	21.4	515	40.2	350	27.6

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[*] Shekelle RB, Dyer AR, Stamler J: Coffee, serum total cholesterol and risk of coronary heart disease in a population of employed, middle-aged men: The Western Electric Study. Presented at a Workshop on Coffee, Plasma Lipids and Coronary Heart Disease, Goteborg, Sweden, May 8–10, 1989.

TABLE 2—Age and Sex-Adjusted Means and Standard Errors of CHD Risk Factors and Proportions of Smokers, Exercisers, and Estrogen Users by Coffee Consumption, Rancho Bernardo 1984–87

	No Coffee mean (se) (n = 249)	Decaffeinated Only mean (se) (n = 439)	Caffeinated Only mean (se) (n = 979)	Both mean (se) (n = 637)
Age (sex-adjusted)	68.94 (.590)	71.79 (.444)	70.15 (.298)	70.68 (.369)
Obesity (kg/m²)	24.99 (.230)	25.39 (.175)	25.11 (.116)	25.09 (.144)
Alcohol (# drinks in past week)	4.08 (.495)	6.19 (.374)	7.08 (.250)	5.99 (.309)
Dietary Fat and Cholesterol index	33.97 (.388)	34.02 (.293)	35.86 (.195)	35.26 (.242)
Night Sleep (hours)	7.17 (0.79)	7.20 (.059)	7.27 (0.40)	7.23 (.050)
Smokers (% current)	5.40 (8.71)	11.16 (13.0)	15.05 (14.9)	11.69 (13.0)
Exercisers (% current)	82.49 (35.1)	82.07 (34.6)	80.23 (34.4)	80.91 (34.5)
Estrogen (women only)	26.34 (27.2)	34.11 (31.0)	27.17 (28.2)	27.54 (27.8)

TABLE 3—Age and Sex-Adjusted Means and Standard Errors of CHD by Consumption of Caffeinated Coffee, Rancho Bernardo, 1984–87

	Cups per Day			
Mean (SE)	1–2 (n = 216)	3–4 (n = 447)	≥5 (n = 953)	p-value (trend)
Age (sex-adjusted)	71.49 (.293)	69.62 (.428)	67.01 (.617)	<.01
Obesity	24.89 (.116)	25.53 (.169)	25.45 (.246)	.04
Alcohol (past week)	6.44 (.261)	7.08 (.380)	7.14 (.552)	.26
Dietary Fat and Cholesterol index	35.29 (.198)	36.46 (.288)	35.61 (.419)	.49
Night Sleep (hours)	7.24 (.041)	7.26 (.059)	7.32 (.086)	.42
Smokers (% current)	10.19 (12.3)	15.31 (15.11)	23.73 (19.2)	<0.1
Exercisers (% current)	82.38 (34.9)	80.27 (34.5)	72.45 (33.1)	<.01
Estrogen (women only % current)	25.80 (27.0)	30.63 (30.0)	25.63 (28.3)	.06

beverages, particularly coffee, showed a negative correlation with treadmill exercise capacity which persisted after cigarette smoking and other variables were partitioned out.

Coffee consumption could be an important link in the association between hours of nightly sleep and mortality. Karacan, *et al.*,[22] reported sleep disturbances in coffee drinkers while several other investigations of health-related practices have addressed the association between reported hours of nightly sleep and subsequent all cause mortality.[23–26] In this cohort, hours of nightly sleep were no different in coffee drinkers compared to non-drinkers and sleep hours did not vary with amount of caffeinated coffee consumed in either men or women. It is possible that older adults who found that caffeinated coffee disturbed their sleep patterns had discontinued its use.

In this cohort, 44 percent of men and 49 percent of women drank at least one cup of decaffeinated coffee per day. Behavior patterns in those who drank only decaffeinated coffee were more similar like those of caffeinated coffee drinkers than non-coffee drinkers. These findings suggest that decaffeinated coffee consumption should be considered in studies of the relationship beween coffee drinking and disease whenever a significant proportion of the study population drinks decaffeinated coffee.

In this study, coffee drinking was determined at the same time as all other behaviors, although the period of use for different behaviors varied from current to usual. Others have shown that current diet is a strong predictor of past or usual diet,[27] and we have found a strong correlation between usual and recent alcohol intake in this cohort. It seems unlikely that differences in the period of use explain the observed associations, unless one postulates that drinkers of coffee (with or without caffeine) have a better memory for unhealthy behavior than non-drinkers.

The diet data reported here were not directly validated; we know of no way to confirm dietary fat and saturated/unsaturated fat ratios short of direct observation or a fat biopsy.[28] Indirect validation of both food frequency and 24-hour diet recall was obtained in this cohort by demonstrating expected diet-disease associations. We have reported the expected cross-sectional associations of sodium, calcium, and potassium with blood pressure,[29,30] and inverse associations of fiber with heart attack;[31] potassium with stroke[32] and calcium with hip fracture in prospective studies.[33] Other attributes were also indirectly validated: smoking was inversely associated with HDL and directly with waist-hip-ratio;[34] alcohol was associated directly with waist-hip-ratio, aspartate aminotransferase and HDL;[35] exercise was positively associated with HDL and inversely associated with obesity, pulse rate, and plasma insulin.[36] Any misclassification of self-reported behaviors would be expected to reduce these associations, and not have caused them.

Because caffeinated and decaffeinated coffee consumption were both associated with more atherogenic behaviors, studies which seek to demonstrate the relationship between coffee consumption and morbidity or mortality must consider these other potential behavioral risk factors in the coffee-disease association. The concordance of heavy coffee intake with several lifestyles known to be related to heart disease risk implies a high level of potential confoundng in studies linking coffee consumption to hyperlipidemia and/or coronary heart disease.

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Validation of Mothers' Reports of Dietary Intake by Four to Seven Year-Old Children

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Abstract: The validity of mothers' recall of four to seven year-old children's diet was assessed among 46 first generation Latino immigrant families from the Dominican Republic by comparing intake recalled by the mother to unobtrusive home observations of children. Correlations were moderate to high for calories and for most nutrients. There were no differences in mean intake of total calories or in intake of most macronutrients and micronutrients assessed. At least two-thirds of the children in the lowest (or highest) quintile based on home observation were correctly classified into the lowest or second lowest (or highest) quintiles based on mother's reports for calories and most nutrients. For all food items that were both observed and reported, 51 percent of reported portion sizes were equivalent to observed portion sizes, 15.5 percent were smaller, and 33.5 percent were larger. There was fair to good agreement on the number of food items eaten, with the exception of vegetables. Mothers' recall appears to be useful for classifying children by intake of calories, macronutrients and micronutrients, but provides a somewhat less accurate measure of actual foods eaten, portion sizes, and nutrient levels consumed. (*Am J Public Health* 1990; 80:1314–1317.)

Introduction

Valid measurement of diet is essential to improve understanding of the role of diet in the etiology of diseases and for evaluating dietary interventions. The 24-hour dietary recall is probably the most widely used instrument in surveys and studies of food intake in the United States,[1–7] and its validity has been investigated in adults by comparison with weighed intakes,[8,9] direct observation,[10–12] and other dietary measurement methods.[13–15] Two recently published studies[16,17] have reported on the validity of recall by adults who cook and serve children's meals, usually mothers. No studies of this kind have been conducted in Latino families with young children or have used unobtrusive observation of children in their homes as a validation criterion. Nor have any previous studies assessed the validity of parental recall of children's nutrient intake using regression methods to adjust for caloric intake, permitting estimation of nutrient values that remove the effect of caloric intake.

We assessed the validity of mothers' recall of four to seven year-old children's dietary intake of total calories and 17 nutrients and of portion sizes and food items aggregated within food groups among first generation Latino immigrant families from the Dominican Republic by comparing intake recalled by the mother to unobtrusive home observations of children.

Methods

Subjects and Setting

Subjects were drawn from a sample of four to seven year-old children and their mothers participating in the Columbia University Study of Childhood Activity and Nutrition, a longitudinal study of young children. The total sample comprises families residing in a primarily Latino, densely populated, low-income neighborhood in northern Manhattan, New York City. Eligibility required that children be generally healthy with no special dietary prescriptions or restrictions.

A total of 46 Dominican families (mean age of children = 5.4 years, S.D. = 0.8) participated in the validation study. There were 18 males (mean age = 5.0 years, S.D. = 0.78 years) and 28 females (mean age = 5.7 years, S.D. = 0.78 years). All parents in the validation study were mothers (mean age = 33.8 years, S.D. = 4.6). Thirty-one of the children lived in single parent families.

Measurements

Home observation: Home observations were conducted between May 1987 and May 1989 by trained observers, fluent in Spanish, on the day before a regularly scheduled study visit in which the mother was to report her child's dietary intake for the preceding 24-hour period. The time period of the home observation ensured observation of the main meal (usually 4:00pm to 8:00pm). Parents were told that the observer was interested in the child's activities but were not informed about the specific aims of the study.

The observer followed the child and recorded the foods the child ate, the amounts, and the time. When necessary the observer asked the child's mother what the child was eating. Prior to data collection, observers were trained and tested in visually estimating portion sizes. Data were recorded on an instrument adapted from the Health and Nutrition Examination Survey.[7]

Twenty-four hour dietary recall: Different individuals conducted the interviews and home observations. On the day following the home observation the mother was asked to describe the food items eaten by the child during each eating occasion, the time of day, and portion sizes. Three dimensional food models, food packages, glasses, cups, spoons, and other utensils, and questions about food preparation methods and brand names of foods were used to increase accuracy. The sections of the recall corresponding to the time when the observer was present in the home were excerpted for analysis. For both observed dietary intake and 24-hour recall data, foods and portion sizes were converted to nutrient intakes using a dietary analysis program,[18] which used published nutrient data from the United States Depart-

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ment of Agriculture.[19,20] Supplemental nutrient values were obtained from other sources.[21]

Statistical Analysis

Nutrient intake values were adjusted for average caloric intake using regression techniques.[22] The natural logarithm transformation was used to improve normality for intake of nutrients but untransformed values are reported in the tables. Nutrient analyses were also performed using unadjusted values. Results were the same and only adjusted values are reported. Pearson product-moment correlation coefficients were used to estimate consistency between observed and reported calorie and nutrient intake. Paired t-tests were used to evaluate differences between means. We also examined the proportion of children correctly classified into quintiles of nutrient intake.[22] Consistency between observed and reported portion size was calculated within nine food groups comprising the 200 food items that were both observed and reported. Portion sizes were defined as equivalent if they were within one and one-half times the smaller amounts. The proportions of mothers who reported fewer, the same, or more items than observed within food groups were calculated, and the Z test[23] was used to assess differences in proportions.

Results

Correlations were moderate to high for most nutrients, although mean nutrient intakes derived from mothers' reports tended to be higher than those derived from observations (Table 1). Paired t-tests indicated no significant differences for total calories or macronutrients, except protein. For micronutrients, there were differences for cholesterol, calcium, iron, thiamine, riboflavin, and niacin. At least two-thirds of the children in the lowest quintile based on home observation were correctly classified into the lowest or second lowest quintiles based on mother's reports for all 17 nutrients except carbohydrates, calcium, and riboflavin (Table 2). Fewer children were correctly classified into the highest two quintiles for fats and cholesterol. For micronutrients, mother's reports were about as good as or better at high levels of intake than they were at low levels of intake. For all nutrients, mean observed intake in the highest quintile was at least three times as high as that in the lowest quintile, so that correct classification has practical significance.

Overall, for all 200 food items that were both observed and reported, 51 percent of reported portion sizes were equivalent to observed portion sizes, 15.5 percent were smaller, and 33.5 percent were larger (Table 3). When mothers made errors in reporting portion sizes they tended to overestimate for all food groups except butter/fats/oils. For all foods combined the proportion underestimated was significantly lower than the proportion over-estimated. The small numbers of dairy products (n = 4) and eggs consumed (n = 1) precluded meaningful analysis for these food groups.

There was fair to good agreement on the number of food items eaten, with the exception of vegetables (Table 4). In all nine food groups mothers tended to report fewer items eaten than observed. The practical significance of the percent agreement depends in part on the overall frequency of consumption. The proportion of mothers who agreed with the observer that items were not eaten is thus included in Table 4. For nine out of 10 of the most frequently eaten foods, observed and reported frequencies were identical (data not shown).

Discussion

Ability to measure children's absolute nutrient intake (e.g., 300 milligrams of cholesterol per day) as well as macronutrient intake as a proportion of absolute calories is important since some nutritional guidelines are stated in these terms.[24–26] Adjustment for caloric intake is necessary when assessing the association between particular nutrients and disease risk or the causal role of nutrients in disease processes.[27–29] The regression method of adjustment recommended by Willett and used in this study[22] permits the estimation of nutrient values while removing the effect of total caloric intake. This is not achieved by dividing nutrient values by total caloric intake.[30]

TABLE 1—Means, standard deviations (SD), Pearson correlation coefficients, and 95 percent confidence intervals for observed and reported nutrients and calories (data based on reports from 46 Dominican mothers residing in Northern Manhattan and corresponding home observations of children's diet, 1987–1989)*

	Observed mean		Reported mean			
	Mean	(SD)	Mean	(SD)	r	95% CI
Fat (g)	12.2	(5.4)	12.9	(7.1)	.52	(.27–.70)
Saturated fat (g)	3.0	(2.5)	3.3	(2.1)	.38	(.10–.60)
Monounsaturated fat (g)	4.1	(2.6)	4.0	(2.3)	.41	(.14–.63)
Polyunsaturated fat (g)	3.9	(2.6)	4.1	(3.0)	.58	(.35–.75)
Cholesterol (mg)	53.6	(84.6)	83.2	(230.5)	.65	(.44–.79)
Protein (g)	16.1	(7.1)	19.7	(8.0)	.50	(.25–.69)
Carbohydrates (g)	59.9	(14.3)	59.8	(16.0)	.48	(.22–.68)
Sodium (mg)	335.9	(302.0)	396.6	(698.4)	.27	(−.02–.52)
Potassium (mg)	566.5	(280.4)	656.9	(546.7)	.54	(.30–.72)
Calcium (mg)	63.0	(57.2)	90.6	(110.7)	.28	(−.02–.52)
Phosphorus (mg)	185.5	(64.9)	230.8	(108.3)	−.10	(−.38–.20)
Iron (mg)	3.0	(1.1)	3.6	(1.3)	.82	(.70–.90)
Vitamin A (IU)	1121.6	(1561.2)	2354.4	(5933.5)	.66	(.45–.80)
Thiamine (mg)	0.28	(0.11)	0.32	(0.12)	.68	(.48–.81)
Riboflavin (mg)	0.22	(0.10)	0.36	(0.43)	.52	(.26–.70)
Niacin (mg)	4.1	(2.2)	4.7	(2.3)	.72	(.55–.84)
Vitamin C (mg)	33.0	(37.3)	29.2	(29.3)	.50	(.25–.69)
Calories	465.0	(229.5)	507.0	(258.9)	.71	(.53–.83)

*Nutrient intakes were adjusted using residuals from regression models with caloric intake as the independent variable and nutrient intakes as the dependent variables (22).

TABLE 2—Comparisons of observed and reported nutrient and caloric intake based on joint classification by quintiles (data based on reports from 46 Dominican mothers residing in Northern Manhattan and corresponding home observations of children's diet, 1987–1989)*

	Joint classification by quintiles					
	Lowest quintile on observation			Highest quintile on observation		
	Lowest quintile on recall (%)	Lowest 2 quintiles on recall (%)	Highest quintile on recall (%)	Highest quintile on recall (%)	Highest 2 quintiles on recall (%)	Lowest quintile on recall (%)
Fat (g)	56	89	0	78	89	11
Saturated fat (g)	33	67	0	63	63	13
Monounsaturated fat (g)	67	100	0	50	63	0
Polyunsaturated fat (g)	44	100	0	44	67	11
Cholesterol (mg)	67	100	0	11	44	22
Protein (g)	44	67	11	67	89	0
Carbohydrates (g)	44	56	11	56	100	0
Sodium (mg)	33	67	0	56	89	11
Potassium (mg)	44	78	0	44	67	0
Calcium (mg)	44	56	22	67	78	11
Phosphorus (mg)	56	67	11	56	78	0
Iron (mg)	80	90	0	56	89	0
Vitamin A (IU)	44	89	0	56	100	0
Thiamine (mg)	56	89	0	44	78	0
Riboflavin (mg)	44	56	11	56	78	0
Niacin (mg)	67	100	0	44	89	0
Vitamin C (mg)	56	78	0	56	67	11
Calories	67	100	0	44	89	0

*Nutrient intakes were adjusted using residuals from regression models with caloric intake as the independent variable and nutrient intakes as the dependent variables (22).

TABLE 3—Portion size reported versus observed for the 200 food items both observed and reported by food group (data based on reports from 46 Dominican mothers residing in Northern Manhattan and corresponding home observations of children's diet, 1987–1989)*

Food Group	Number of Foods	% Observed > Reported	% Observed = Reported	% Observed < Reported
Eggs	1	0	0	100.0
Dairy products	4	0	75.0	25.0
Butter/fat/oil	13	46.2	30.8	23.1
Sugar	17	11.8	58.8	29.5
Meat/fish/poultry	23	13.0	52.2	34.8
Fruits	22	9.1	59.1	31.8
Beans/nuts	36	16.7	38.9	44.4
Bread/grains/rice/cereal	40	15.0	52.5	32.5
Vegetables	44	13.6	56.8	29.5
Total**	200	15.5	51.0	33.5

*Portion sizes were defined as equivalent if they were within one and one-half times the smaller amount.

**Difference in proportions overreported − underreported = .18 (95% confidence intervals .08–.27)

Our findings support the utility of mother's recall of children's diet for research needing accurate classification of subjects' nutrient intake according to an ordinal or interval level of measurement. The differences in mean levels of intake for protein and several of the micronutrient values raise concerns about the use of parental recall for testing differences in mean nutrient intake between groups. Our results also indicate substantial errors in estimating portion sizes of foods, particularly over-estimation, as well as overreporting and underreporting of specific food items, and thus raise questions about the use of mother's recall as a method of measuring these aspects of children's eating behavior. Studies that require precise and accurate estimates of mean nutrient intake within groups or descriptions of food items and portion sizes consumed may need to rely on direct observation.

TABLE 4—Percent of respondents reporting fewer, the same or more food items (vs observed) by food group (data based on reports from 46 Dominican mothers residing in Northern Manhattan and corresponding home observations of children's diet, 1987–1989)

	Items Reported (vs Observed)			
Food Group	% Fewer	% Same	% More	% Agree not Eaten
Eggs	4.3	95.7	0	93.5
Dairy products	8.7	84.8	6.5	76.1
Butter/fat/oil	21.7	71.7	6.5	39.1
Sugar	17.4	69.6	13.0	37.0
Meat/fish/poultry	15.2	73.9	10.9	34.8
Fruits	19.6	69.6	10.9	45.7
Beans/nuts	15.2	73.9	10.9	28.3
Bread/grains/rice/cereal	10.9	80.4	8.7	15.2
Vegetables	30.4	41.3	28.3	17.4

The dietary variable of interest in chronic disease epidemiology is typical diet. Eating patterns vary by time of week and year,[31–33] so that a single 24-hour dietary recall will not represent typical diet. Mothers' recall of children's evening meal may differ from that of other meals, and the presence of the observer may influence mothers' menu for the day or her likelihood of remembering what she served. The main evening meal is important, however, as evidenced by the observed consumption of calories, fat and saturated fat, as well as other nutrients.

Results from this study are consistent in some respects with other reports of the utility of surrogate-reported dietary information. The studies in adults, which reached guarded conclusions about the utility of this strategy, assumed that subjects' reports were accurate and these reports were the criterion against which the surrogates' reports were tested.[34–36] A trained observer provided the criterion measure in our study. Klesges, *et al*,[16] performed home observations of 30 children ranging in age from 24 to 48 months in which the observer weighed and recorded all food eaten by the child during the day. Dietary recalls were taken from the parent later that same day. At the nutrient level findings were similar to those in our study. At the food level, Klesges, *et al*, found that parents made errors in estimating portion sizes but correctly recalled 96 percent of the food items the child ate, a level of accuracy higher than in our study. Collecting recall data from the parents on the same day as the observation, rather than on the day following the home observation (which is how the recall method is generally used), or having the observer weigh food prior to consumption by the child may have enhanced the accuracy of reporting.

Eck, *et al*,[17] conducted unobtrusive observations of 34 children four to nine years of age eating a single meal in a cafeteria with both of their parents. Dietary recalls were taken from family members individually and as a group on the following day. There were no significant differences between observed and reported mean intake of total calories, calories from total fat, protein, carbohydrates, or sugar, or in intake of cholesterol, sodium, iron or calcium. The mean correlation for these nutrients was 0.77 for fathers, 0.63 for mothers, and 0.86 for the mother, father and child as a group. Eck, *et al*, also examined overreporting and underreporting of nutrients by food groups and reported that while general agreement was high, over- and underreporting varied greatly. The correlations between reported and observed nutrient intakes were, in general, higher than in our study. This study was based on a single meal in a cafeteria setting eaten by middle to high socioeconomic status families,[17] while our study was done in the home of Latino subjects of lower educational attainment.

Our study, together with the investigations by Klesges, *et al*, and Eck, *et al*, indicate that mothers' reports of children's diet appear to be useful to measure children's intake of calories, macronutrients, and micronutrients. The recall gives a somewhat less accurate measure of actual foods eaten, portion sizes, and nutrient levels consumed. Additional research is needed to verify our results in other Latino subgroups and non-Latino populations, to improve understanding about factors that improve and impede mother's reports about children's diet, and to test strategies to improve the validity of these measures.

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Development and Validation of a Food Use Checklist for Evaluation of Community Nutrition Interventions

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Abstract: We developed a new dietary assessment instrument, the Food Behavior Checklist (FBC), which measures food use related to adopting lower-fat and higher-fiber diets. The FBC is a simplification of the 24-hour diet recall that consists of 19 simple yes/no questions about foods consumed during the previous day. To develop the FBC, an expert committee generated a list of foods based on our intervention program, and we used focus groups and random-digit dialing pretests to refine the format and clarify items. To validate the FBC, we compared responses of 96 women on the FBC to information collected during a professionally administered 24-hour diet recall. For most items, agreement between the FBC and 24-hour recall, based on the kappa statistic, was good to excellent. Agreement was poor for items requiring detailed knowledge about food composition (e.g. high-fiber cereal). There was a trend to over-report general food categories (e.g. luncheon meats) but not specific food items (e.g. ice cream). In an embedded randomized study, we found that a set of introductory items designed to serve as a memory retrieval cue did not improve agreement between the FBC and 24-hour recall. These data provide preliminary evidence supporting the use of short checklist questionnaires on the previous day's food use as a means to assess diet at the group or community level. This relatively inexpensive and rapid measure can be used to inform the design of public health nutrition programs and as an evaluation tool in intervention research. (*Am J Public Health* 1990; 80:1318–1322.)

Introduction

There is an emerging consensus that dietary change interventions are a necessary component of public health programs to prevent cardiovascular diseases and cancer.[1] Although the details of recommendations differ, the principal dietary goals shared by leading American public health organizations[1–3] are to: 1) reduce fat intake from approximately 37 to 30 percent or less of total calories; and 2) increase the intake of nutrient- and fiber-rich fruits and vegetables from approximately two to five or more servings per day. Much recent research has focused on community-level interventions, such as mass media campaigns and supermarket programs, because public health professionals believe that these interventions will be the most cost-effecitve means to change dietary behavior.[4]

Both the design and evaluation of public health nutrition intervention programs have problems in assessing dietary behavior in target communities. Standard diet assessment instruments used for clinical and etiological research such as multiple-day diet records and food frequency questionnaires are lengthy, expensive, and difficult to administer to representative population samples. Even the 24-hour recall, the traditional survey interview method used to assess group-level dietary intake,[5] is problematic in community intervention research; it takes approximately 30 minutes to complete, it is sufficiently complex to require both professionally trained interviewers and rigorous standardization and analysis procedures, and it is expensive to administer, code, and analyze. Clearly, new approaches to dietary assessment are needed.

Research on dietary assessment methods appropriate for community-level nutrition intervention research has focused on three approaches. The first approach is based on ecological-level measures: supermarket warehouse orders,[6] supermarket sales,[7] supermarket characteristics (such as percent of meat cases offering chicken, turkey, and fish[8]) and cafeteria receipts.[9] A second approach is based on short dietary questionnaires designed to assess only a limited number of nutrients.[10,11] A third approach is based on questionnaires that assess only the dietary behavior targeted by the intervention program. Several examples of these instruments have been described, for example the MRFIT fat counter,[12] but little is known about the statistical characteristics of these measures when used as program evaluation tools.

In this report we describe a new instrument to assess group-level dietary behavior that is based on the approach of assessing specific target behavior, the Food Behavior Checklist (FBC). The FBC is a simplification of the 24-hour recall, consisting of simple yes/no questions about foods consumed in the previous day. It focuses on a recent and defined time period and may thus reflect more accurately true food use compared to recall of perceived "usual" diet.[13] The advantage of the FBC over the traditional 24-hour recall is that the FBC can be administered quickly by non-professional personnel, needs no coding before analysis, can be completed over the telephone, and can be easily tailored to a specific intervention program. It was developed originally as a component of the evaluation of the Nutrition and Cancer Prevention Program of the California Department of Health Services. The FBC was subsequently incorporated into the evaluation of the Kaiser Family Foundation's Community Health Promotion Grants Program.* Here we describe the instrument, its development, and a study to evaluate its validity.

Methods

Development of the Questionnaire

The goal of the Nutrition and Cancer Prevention Program is to change the consumption of foods which are

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* Wagner EH, Kehrer B. The Henry J. Kaiser Family Foundation Community Health Promotion Program. Evaluation design considerations. Presented at the annual meeting of the International Epidemiological Association, Helsinki, Finland, August 1987.

believed to contribute to the development of cancer—to increase the use of foods containing protective nutrients (vitamin C, carotenoids, and dietary fiber) and decrease the use of foods high in fat. The program goals were operationalized as: increasing the consumption of fruits, vegetables, whole grain/high fiber products and reduced-fat milk, and decreasing the consumption of full-fat dairy products, fried foods, ground beef and other high-fat meats, pastries, cakes, ice cream, and butter or margarine on bread and vegetables. We used these intervention goals to generate a list of foods for inclusion in the FBC.

The draft FBC was reviewed by an expert panel, including epidemiologists, nutritionists, a statistician, and a cognitive psychologist. A series of pre-tests was completed over the telephone, using respondents from a broad range of socioeconomic and age groups. Based on the results of these steps, an introductory section asking the respondent to recall which meals and snacks were consumed the previous day was added, several items were eliminated, and the questionnaire was reworded and reordered.

We then used focus groups to examine the relevance of the instrument to different ethnic groups (Black, Hispanic and Asian) and to men as well as women. The questionnaire was again reworded, and several ethnically specific foods were added as descriptions to the original food behavior items. Finally, we completed a full-scale, random-digit dial pilot survey of 299 adult residents of two California communities. From this pilot we compiled a final interviewer manual and code book (available from the authors) and completed minor rewording. The 19 items included in the instrument are given in the Appendix.

Validation Study

Detailed descriptions of the sampling scheme, participant demographic characteristics, and dietary assessment methods used in this study have been published.[11-14] Briefly, participants were 96 women ages 45 through 59 years who were members of a large health maintenance organization in Western Washington State. Dietary assessments included two, four-day diet records (at study entry and six months later) and a food frequency questionnaire (two months after study entry). Participants received no information about nutrition or their diets until all data were collected.

The validity study was completed by telephone three months after study entry. The interview consisted of: the FBC; a four-minute set of questions on usual dietary behavior; and a detailed, standardized 24-hour recall administered by a research nutritionist (HH or AS). The FBC used in the validation study differed slightly from the final FBC because pretesting had not yet been completed. These differences are given in the Appendix. The 24-hour recall was administered by working the participant through their previous day, using prompts to obtain a complete list of foods and asking questions to get specific information (brand of cereal, type of bread, etc). Nutritionists defined a high-fiber cereal as one with at least 3g of dietary fiber per serving; whole grain breads included all breads with the exception of white breads and caramel-colored breads made without whole grain flour. We considered this 24-hour recall to be a good criterion measure of foods eaten in the previous day because 24-hour recalls are the most commonly used method to obtain short-term, retrospective data on dietary intake.[5]

We also considered whether the use of introductory questions on the context of the previous day's food consumption would improve the validity of responses to the FBC. Studies of human memory have shown that context can serve as an effective retrieval cue for memories of personal events.[15] Furthermore, studies of survey respondents indicate that many persons first think about the circumstances surrounding a health event when they are asked to specify its date or frequency.[16] This suggests that specific, incidental events such as eating a bowl of cereal may be better remembered if the individual first thinks of the more easily remembered context such as having breakfast. Study participants were thus randomized into two groups: receiving or not receiving five introductory questions before beginning the FBC. These introductory questions were similar to probes used during a 24-hour recall, and asked about whether the participant ate breakfast, lunch, and dinner, whether they ate in a restaurant and, if so, in what type of restaurant (fast food/other).

Statistical Methods

We used two approaches to measure agreement between the FBC and the criterion measure, the 24-hour recall.

- The first approach was to compare the proportion of participants who reported each food behavior from the FBC to the proportion reporting the same behavior during the 24-hour recall. This comparison considers whether FBC and 24-hour recall would yield different community-level estimates of dietary behavior practiced in the previous day. We calculated confidence intervals around these differences using the exact binomial distribution.[17]
- The second approach to assess validity was to examine the proportion of individuals with the same responses from the FBC and the 24-hour recall. This analysis considers whether the FBC and 24-hour recall differ when measuring the behavior of individuals. We used the kappa statistic to assess agreement in this application.[18] In presenting the results, we indicated items for which expected chance agreement was over 90 percent because the kappa statistic in this case will be based on very few discordant pairs and thus be highly unreliable.[19]

Finally, we examined the association of items on the FBC with usual percent of energy from fat and usual fiber intake. To characterize usual dietary intake in these analyses we used the average of values from the food frequency questionnaire and eight days of diet records. We did not expect that all items on the FBC would be strongly associated with usual dietary intake because the use of a food in the previous day is not a reliable measure of usual food use.[5] However, we considered associations of FBC items with usual dietary intake as evidence of the content validity of the instrument.

Results

Table 1 gives the proportion of participants reporting use of 18 foods in the previous day from both the FBC and the 24-hour recall. Overall, the proportion of yes responses tended to be higher for the FBC than the 24-hour recall (sign test $p < 0.01$), although the 95 percent confidence intervals for these differences excluded zero for only five items: high-fiber cereal, fruit, a vegetable at lunch, a vegetable at dinner, and whole-grain bread. The percent of concordant responses and the corresponding kappa statistic for each item are also given in Table 1. Observed agreement was under 85 percent for only three items: high-fiber cereal; whole-grain breads; and butter or margarine on bread. Only two kappas, for high-fiber cereal and bacon or sausage, were under 0.60,

TABLE 1—Agreement between Food Behavior Checklist and 24-Hour Recall

Food Behavior Checklist Item*	Responded Yes: Food Behavior Checklist (%)	Responded Yes: 24-Hour Recall (%)	95 Percent Confidence Interval for Difference	Agreement: (%)	Agreement: Kappa
Cereal	50.0	50.0	(−1.9, 1.9)	97.9	0.96
High-fiber cereal	89.1	65.2	(12.0, 23.9)	76.1	0.37
Pastry	13.5	9.4	(0.0, 4.2)	95.8	0.80
Bacon or sausage	3.1	4.2	(−3.0, 2.3)	96.9	0.56**
Milk	65.3	65.3	(−0.2, 0.2)	91.6	0.81
Fruit	90.5	84.2	(1.2, 6.3)	93.7	0.72
Salad	52.1	45.8	(−6.8, 10.2)	85.4	0.71
Vegetable at lunch	34.4	24.7	(0.0, 13.1)	86.0	0.67
Vegetable at dinner	68.4	53.7	(8.7, 14.7)	85.3	0.70
Butter on vegetable	19.0	12.6	(−3.9, 9.6)	89.5	0.61
Dark bread	56.3	45.8	(2.4, 14.9)	83.3	0.67
Muffins	17.7	15.6	(−3.5, 6.5)	91.7	0.70
Butter on bread	37.5	33.3	(−4.9, 10.7)	83.3	0.64
Fried food	7.3	7.3	(−1.9, 1.9)	97.9	0.85
Cold cuts	6.3	6.3	(−2.3, 2.3)	95.8	0.64
Ground beef	13.5	13.5	(−2.3, 2.3)	95.8	0.82
Cakes	45.8	45.8	(−2.3, 2.3)	95.8	0.92
Ice Cream	14.6	14.6	(−1.9, 1.9)	97.9	0.92

*Full text of items in Appendix.
**Expected chance agreement >90%.
N = 96

a level that may be considered a cutpoint for fair agreement beyond chance. For the remaining 16 items, agreement was very good to excellent for 11 (kappa > 0.80) and good for five (kappa > 0.60).

In further analysis stratified by whether or not participants received the introductory questions to the FBC, there was no evidence that introductory questions improved the quality of responses (data available from the authors). The tendency for a higher proportion of participants to report food use in the FBC than in the 24-hour recall remained evident in both strata. This bias was not smaller among participants who received the introductory questions, nor was there a trend for kappas to be higher.

The associations of usual dietary intakes of fat and fiber (assessed from a food frequency and eight days of dietary records) with responses to the FBC are given in Table 2. With only a single exception (vegetable at dinner) the direction of association was consistent with the fat or fiber content of each food. Confidence intervals for differences in percent of energy from fat excluded zero for consumption of pastry, salad, butter or margarine on breads, and cold cut luncheon meats. Confidence intervals for fiber intake excluded zero for only cereal and fruit. These results did not differ when food use in the previous day was taken from the 24-hour recall.

Discussion

Overall, reports of previous day's food behavior related to fat and fiber intake appeared similar when assessed by either the Food Behavior Checklist or a 24-hour recall. There

TABLE 2—Mean Usual Percent of Energy from Fat and Fiber Intake, by Response to Food Behavior Checklist

Food Behavior Checklist Item*	Percent of Energy from Fat Response to FBC: "Yes"	Percent of Energy from Fat Response to FBC: "No"	95 Percent Confidence Interval for Difference	Fiber (g) Response to FBC: "Yes"	Fiber (g) Response to FBC: "No"	95 Percent Confidence Interval for Difference
Cereal	31.8	33.2	(−3.9, 1.0)	18.2	16.1	(0.0, 4.1)
High-fiber	31.9	31.5	(−5.7, 6.4)	18.6	15.3	(−1.4, 8.0)
Pastry	35.8	32.0	(0.2, 7.3)	16.7	17.2	(−3.6, 2.6)
Bacon or sausage	37.5	32.3	(−1.8, 12.2)	15.5	17.2	(−7.8, 4.4)
Milk	32.4	32.6	(−2.8, 2.4)	17.5	16.4	(−1.1, 3.4)
Fruit	32.3	34.9	(−6.8, 1.6)	17.6	13.2	(0.8, 7.9)
Salad	30.3	34.9	(−6.9, −2.3)	17.7	16.5	(−0.9, 3.3)
Vegetable at lunch	31.6	32.8	(−3.8, 1.5)	18.2	16.4	(−0.4, 4.1)
Vegetable at dinner	32.0	33.6	(−4.2, 1.1)	16.8	17.7	(−3.1, 1.4)
Butter on vegetable	34.6	32.0	(−0.5, 5.7)	17.4	17.1	(−2.5, 3.0)
Dark bread	31.8	33.4	(−4.1, 0.8)	18.0	16.1	(−0.2, 4.0)
Muffins	30.9	32.8	(−5.1, 1.3)	18.0	16.9	(−1.7, 3.8)
Butter on bread	34.4	31.4	(0.6, 5.5)	17.1	17.2	(−2.6, 2.4)
Fried food	34.2	32.4	(−2.9, 6.6)	16.5	17.2	(−4.8, 3.3)
Cold cuts	39.4	32.0	(2.5, 12.2)	14.3	17.3	(−7.4, 1.3)
Ground beef	34.2	32.2	(−1.6, 5.6)	15.9	17.3	(−4.5, 1.7)
Cakes	33.0	32.1	(−1.6, 3.4)	16.1	18.0	(−3.9, 0.3)
Ice cream	35.0	32.1	(−0.5, 6.4)	16.9	17.2	(−3.2, 2.7)

*Full text of items in Appendix.

was a general trend for overreporting food use on the FBC, but this did not appear to be a bias to report more socially desirable behavior. Both desirable (e.g. "vegetable at dinner" and "high-fiber cereal") and undesirable (e.g. "butter or margarine on bread" or "breakfast or snack pastry") behavior were reported more frequently on the FBC than the 24-hour recall.

We suspect that forward telescoping,[20] a term used in cognitive psychology to describe recall of a past event as more recent in time, may explain higher responses from the FBC. In this study, we observed telescoping for common or non-specific dietary behavior, such as vegetable and fruit eating; responses to more specific dietary behavior, such as use of cold cuts or ice cream, were identical from the FBC and 24-hour recall. This suggests that a checklist approach to dietary assessment should emphasize specific rather than general dietary behavior. For example, it may be better to read a list of specific vegetables or to ask "What vegetables did you eat with lunch" rather than to ask simply "Did you eat a vegetable with lunch?"

From a cognitive perspective, the FBC has a number of advantages over more complex instruments to assess dietary behavior. First, the question format is straightforward. This feature reduces variation in interviewer behavior and simplifies the response task of the participant.[21] Second, participants do not appear to find remembering the previous day's food behavior difficult. Finally, the use of a 24-hour period provides the respondent with a simple landmark. For questions on specific dietary behavior this may have reduced the telescoping errors typical of more long-term recall periods.[22]

There was also evidence that questions requiring high levels of nutritional knowledge did not produce valid data. For the questions on high-fiber cereal and whole-grain bread we determined that the source of disagreement between the FBC and 24-hour recall was due to the nutritionists' correct classification of the food based on brand name information obtained during the 24-hour recall. For example, participants may report that they are eating a high-fiber cereal when, based on published nutrient composition data, a serving of the cereal actually contained less than 3 grams of fiber per serving. In the final FBC, the "high-fiber" cereal question was modified to a list of specific high fiber cereals, eliminating the need for the respondent to know the fiber content of cereals they ate.

We had hypothesized that the introductory questions would serve as a memory retrieval cue and improve the accuracy and speed of responses. Contrary to our expectations, interviews with the five simple, introductory questions took an average of 1.4 additional minutes to complete ($p < 0.01$) and resulted in no meaningful improvements in agreement between the FBC and 24-hour recall. These results suggest that the FBC items did not pose a significant challenge to respondents' memory abilities.

Given the characteristics of participants in this study there are limits to generalizing these results to more representative population samples. Participants in this study were all women, interested in diet and health, well educated, and willing to comply with intensive measurement protocols. Evaluation of the FBC in other populations is now under investigation. The FBC has been used successfully during baseline assessments of 15 communities in the Kaiser Family Foundation's Community Health Promotion Grants Program, and we have found strong community-level associations between the FBC and measures of supermarket characteristics.[23] The FBC has also been incorporated into an ongoing random-digit dial health survey of Washington State residents, and validation studies from this survey are underway.

Data collected from the FBC lend themselves to two types of analyses. When data are collected at the beginning of a planned intervention, they can be used to document problems and formulate program goals. When data from an FBC are collected pre- and post-program implementation in intervention and control communities, they can be used to evaluate changes in the prevalence of food use attributable to an intervention program. Thus, if intervention program goals can be clearly formulated as increases or decreases in the prevalence of use of specific foods, the FBC may be a superior instrument to standard dietary questionnaires which attempt to measure total intake of nutrients.

In conclusion, there is preliminary evidence that a checklist questionnaire is a reasonable approach to community-level dietary assessment. We have demonstrated that a careful process of selecting questionnaire items can produce an instrument with both face and content validity, and answers to a checklist questionnaire are similar to those obtained from a 24-hour recall. The FBC may be an inexpensive and technically simple dietary assessment tool when group-level data on dietary behavior can be used to evaluate intervention programs or monitor dietary change.

APPENDIX

Food Items Included in Food Behavior Checklist

Cereal (hot or cold)
  a. If yes, was it a high fiber cereal?[*]
    a. If yes, was it any of the following? Any bran cereal, Raisin Bran, Wheatena, Shredded Wheat, Oatbran, Oatmeal or Fruit n Fiber?[**]
Breakfast or snack pastry such as donuts, croissants, danish, coffee cake, or sweet rolls, pan dulce or custard tarts
Bacon, sausage, or chorizo
Milk, including milk on cereal, chocolate milk, or cafe con leche
  a. If yes, was it[**]
    regular or whole milk?
    low-fat or 2% milk?
    skim, 1% or non-fat milk?
One or more servings of fresh fruit
Green salad
Vegetable, other than salad, at lunch
Vegetable, other than salad, at dinner
Butter, margarine, or creamy sauce on cooked vegetables
Dark bread, including whole wheat, rye, or pumpernickel
Whole grain or bran muffins
Butter or margarine on bread
Deep-fried food like french fried potatoes, fried chicken or fish, tacos, egg rolls, or dim-sum
Hot dog, salami, bologna, or other luncheon meat
Hamburger, meat loaf, tacos or other dish with ground beef
Cake, pie, custard, tart or cookies
Ice cream

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[*] Included in validity study only

[**] Included in final instrument only

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Food and Nutrient Intake Differences between Smokers and Non-smokers in the US

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Abstract: Data from the Second National Health and Nutrition Examination Survey were analyzed to determine food and nutrient intake differences between current smokers (also categorized as light, moderate, and heavy smokers) and non-smokers. Smokers in several age-race-sex categories have lower intakes of vitamin C, folate, fiber, and vitamin A than non-smokers, and intake tended to decrease as cigarette consumption increased, particularly for vitamin C, fiber, and folate. Smokers were less likely to have consumed vegetables, fruits (particularly fruits and vegetables high in vitamins C and A), high fiber grains, low fat milk, and vitamin and mineral supplements than non-smokers. A negative linear trend was found between smoking intensity and intake of several categories of fruits and vegetables. These data suggest that the high cancer risk associated with smoking is compounded by somewhat lower intake of nutrients and foods which are thought to be cancer protective. (*Am J Public Health* 1990; 80:1323–1329.)

Introduction

Cigarette smoking is the biggest single health habit that contributes to preventable chronic disease in the United States especially cancer of the lung, larynx, oral cavity, and esophagus as well as coronary artery disease and emphysema.[1] Other cancers, such as bladder, kidney, pancreas, cervix, and stomach are also associated with cigarette consumption. Cigarette use in the US has been estimated to be a causative factor in 30 percent of all cancer deaths and in 87 percent of all lung cancer deaths.[2] It has also been estimated that diet is a causative factor in 35 percent (with a possible range of 10–70 percent) of all cancer deaths. While smoking, food, and alcohol consumption each are lifestyle practices associated with cancer risk, when they occur together some authors assert they act synergistically to produce risks which are greater than the sum of their parts.[3]

Compared to non-smokers, smokers consume more alcohol and coffee, take more risks, have more sexual partners, exercise less, sleep less, take vitamin and mineral supplements less, and skip breakfast more.[4–11] Furthermore, several of these poor health habits are more likely to occur with increasing average number of cigarettes smoked per day.[8]

Among smokers, those with either higher dietary intakes of vitamin and/or carotene or higher serum carotenoids are at reduced risk for lung cancer,[12–25] and several studies have examined the vitamin A, carotenoid, or energy intake differences between smokers and non-smokers.[26–30] A recent study showed an inverse association between intake of vitamin C and smoking.[31] Only one study[32] of 45–59 year old men enrolled in a prospective heart disease trial in Wales, however, examined total nutrient intake differences between smokers and non-smokers, and smokers were found to have lower intakes of most nutrients.

Understanding the dietary patterns of those who smoke compared to those who do not smoke is important for health professionals seeking to assess disease risk and educate individuals regarding healthy lifestyles. We used data from a large nationally representative survey, the Second National Health and Nutrition Examination Survey (NHANES II),[33] conducted from 1976–80, to determine whether smokers significantly differ from non-smokers in both nutrient and food intake.

Methods

NHANES II used a probability sample of the civilian non-institutionalized US population ages 6 months to 74 years. The complex sample design and sample weights are described in detail elsewhere.[34] Mobile examination centers employing trained teams of physicians, nurses, dietitians, and technicians traveled to and collected data from 64 sites throughout the country.

NHANES II 24-hour dietary recall data from 11,260 adults ages 19–74 years were used to assess dietary intake. For these analyses, races other than Black or White were excluded because of small sample sizes. Also excluded were individuals (N = 280) with imputed, surrogate, or unreliable 24-hours recall data. Trained interviewers collected dietary intake data from the day before the interview. Three-dimensional food portion models were used to help individuals accurately report portion sizes.[35] Dietary data were coded by the interviewers within 72 hours of data collection and analyzed for nutrient content using current nutrient values. Nutrient values for folate and dietary fiber, however, were not available on NHANES II tapes and were added later by Subar, *et al*,[36] and Lanza, *et al*,[37] respectively.

A smoking history was obtained from all subjects. Individuals were placed into categories of current, former, and never smokers, and current smokers were divided into categories of light, moderate, and heavy smokers based upon reported average use of 1–14, 15–24, or ≥25 cigarettes/day, respectively. Initial analyses showed few differences in nutrient or food intakes from 24-hour recall data between never and former smokers and therefore they were combined, creating a group of current non-smokers. Individuals who were not cigarette smokers but smoked either pipes (N = 63) or cigars (N = 140) <6/week, and <3/week, respectively, were included as non-smokers because usage was considered too infrequent to warrant exclusion. Frequent pipe or cigar users (N = 398) were excluded from the analyses.

Nutrient intake differences between current smokers and non-smokers, and among current non-smokers and light, moderate, and heavy smokers in the 24-hour recall data set were examined by race, sex, and broad age groups (19–29, 30–54, and 55–74 years). Differences in food intake were determined using food groups developed by Patterson and Block.[38] The food groups created were based upon the

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interim dietary guidelines by the National Research Council's report, *Diet, Nutrition and Cancer*,[39] and those of the American Cancer Society.[40] An inclusive "all vegetables" category was developed as well as the following vegetable subcategories: cruciferous vegetables (i.e. broccoli, cauliflower, brussels sprouts, cabbage), deep yellow vegetables (carrots, winter squash, sweet potatoes), fiber-rich vegetables (dried peas, beans, corn), and "garden vegetables" (all vegetables except white potatoes, dried peas and beans, and salad). Fruits and vegetables were also subdivided into categories of those high in either vitamin A (i.e. carrots, spinach, cantaloupe) or vitamin C (i.e. oranges, grapefruit, broccoli). A complete listing of foods in each category has been published.[38] Regardless of portion size or numer of servings, individuals were described as having consumed or not consumed food(s) within each food group based upon 24-hour recall data. Preliminary analyses of food group intake indicated that such coding yielded few differences by sex and, therefore, individuals were categorized by age and race only.

In addition to univariate analyses described above, multivariate analyses of nutrient and food intake were performed adjusting for age, kcal/daily, poverty index ratio or PIR (an income status index which takes into account household income, family size, and the income necessary to maintain a family at the time the study was conducted), and sex. These analyses were performed separately for Whites and Blacks because of the known racial differences in consumption of foods and nutrients.[41]

Separate analyses by sex-race categories were performed to determine whether smoking category based upon average daily cigarette consumption, as measured in half packs of cigarettes consumed, was linearly related to intake of nutrients or food groups. Each nutrient or food group, acting as a dependent variable in the model which adjusted for age, examined the effect of half packs of cigarettes consumed. For purposes of these analyses, quartiles of nutrient intake by race-sex groups were used.

Statistical analyses were performed using SAS[42] with appropriate population weights permitting inferences about the total adult US Black and White population. Standard errors were calculated using SESUDAAN,[43] a program designed to account for the complex survey design. The SURREGR[44] program for complex samples was used in multivariate analysis for the White population, but because of the lack of dispersion of Blacks throughout strata and primary sampling units (PSU), an unweighted regression analysis was used for Blacks. Logistic regressions were performed using the RTIlogt program.[45] This application uses the Taylor expansion series to estimate the variances.

Results

Food Group Intake Differences

Analyses by age, race, sex, and current smoking status showed a larger proportion of Black men (55 percent) smoked during the period of the NHANES II study than in any other sex-race category, followed by White men (43 percent), White women (34 percent), and Black women (34 percent). In all age-race-sex groups, approximately 50 percent of all smokers are 30–54 years of age, while approximately one-third and 15 percent are ages 19–29 and 55–74 years, respectively.

Tables 1 and 2 present univariate analyses of food group intake of smokers and non-smokers by age for Whites and Blacks, respectively. With few exceptions, intake of vegetable categories except "starchy fibrous" is moderately higher among non-smokers as compared to smokers in all age-race groups. Multivariate analyses which adjusted for age, kcal/day, PIR, and sex confirmed these results among Whites. Both univariate and multivariate analyses show that non-smokers of both races have a strikingly higher intake of fruits compared to smokers with percentage differences as high as 19 percent (unadjusted) for 19–29 and 30–54 year old Whites, and 55–74 year old Blacks. For the categories of high fiber grains and fruits or vegetables high in either vitamin C or vitamin A, non-smokers in all age-race categories have a greater intake than smokers, a finding confirmed after multivarate adjustment. For high fiber grains, however, it should be noted that Blacks have much lower intakes than Whites with the highest intakes among Black non-smokers equal to or less than the lowest intakes of White smokers.

TABLE 1—Weighted Percent of White Subjects Consuming Food(s) Over a 24-Hour Period (NHANES II) by Age and Smoking Status

	Whites 19–29 years				Whites 30–54 years				Whites 55–74 years			
Food Group	Non-Smoker	95% CI[a]	Smoker	95% CI	Non-Smoker	95% CI	Smoker	95% CI	Non-Smoker	95% CI	Smoker	95% CI
All Vegetables	84.4	82.6,86.1	79.0	76.7,81.3	85.9	84.4,87.4	81.0	79.2,82.9	86.3	84.7,87.9	82.9	79.9,86.0
Garden	49.7	47.0,52.4	42.7	39.3,46.1	54.2	51.4,57.0	48.5	46.4,50.5	58.4	55.9,61.0	51.9	48.3,55.5
Deep Yellow	9.2	7.4,11.1	6.3	4.4,8.1	11.6	9.8,13.4	8.7	6.8,10.6	13.7	12.2,15.2	11.0	8.8,13.2
Cruciferous	15.3	13.4,17.2	12.0	9.9,14.1	20.0	18.5,21.5	15.9	13.4,18.3	21.2	19.3,23.0	18.4	16.3,20.4
Starchy Fibrous	18.8	16.0,21.5	18.8	15.9,21.7	19.1	17.2,21.1	18.2	16.2,20.2	18.0	15.4,20.5	17.9	15.2,20.6
Salad	48.6	45.3,52.0	39.4	35.7,43.0	50.5	48.4,52.6	46.7	43.6,49.9	49.7	47.3,52.1	46.1	42.4,49.8
All Fruits	59.3	56.4,62.3	39.6	35.7,43.5	63.9	61.7,66.1	45.4	42.4,48.5	76.8	74.8,78.7	63.3	60.0,66.5
Fruits/Vegetables												
High in Vitamin A	18.4	15.7,21.1	13.3	11.5,15.1	23.2	21.2,25.2	15.9	13.8,18.1	26.1	24.1,28.1	20.8	17.9,23.7
High in Vitamin C	25.6	22.5,28.7	19.5	16.7,22.3	31.6	29.2,34.1	20.4	17.9,22.9	36.5	33.2,39.7	27.6	24.1,31.1
High Fiber Grains	16.6	13.1,20.0	9.9	6.8,12.9	18.1	15.4,20.9	11.1	8.4,13.7	25.5	23.1,27.9	20.0	16.1,23.8
Red Meat	59.5	56.4,62.6	60.5	57.5,63.4	56.0	53.8,58.3	56.8	54.1,59.5	50.2	48.2,52.1	50.1	46.1,54.1
Poultry/Fish	31.8	29.0,34.6	29.3	26.7,31.9	33.0	30.5,35.5	28.7	25.7,31.8	34.8	32.6,37.1	30.4	26.8,33.9
Bacon/Luncheon Meat	36.2	33.6,38.9	41.2	37.6,44.7	42.4	39.6,45.2	46.6	43.1,50.0	41.4	38.6,44.2	44.9	40.9,49.0
Whole Milk	41.8	38.5,45.0	47.6	43.7,51.4	40.5	37.8,43.1	43.3	39.7,46.9	39.6	36.7,42.6	46.0	41.8,50.3
Low Fat Milk	30.2	26.2,34.3	22.0	18.1,25.8	28.7	25.9,31.6	20.3	17.0,23.7	34.5	31.7,37.4	24.2	20.2,28.3
Use Supplements[b]	24.3	22.0,26.6	15.6	13.3,17.9	24.9	22.4,27.3	20.8	18.0,23.6	34.6	32.1,37.1	29.3	25.9,32.8

a) CI = Confidence interval.
b) Data based upon reported "regular" usage of vitamin/mineral supplements.

TABLE 2—Weighted Percent of Black Subjects Consuming Food(s) Over a 24-Hour Period (NHANES II) by Age and Smoking Status

	Blacks 19–29 years				Blacks 30–54 years				Blacks 55–74 years			
Food Group	Non-Smoker	95% CI[a]	Smoker	95% CI	Non-Smoker	95% CI	Smoker	95% CI	Non-Smoker	95% CI	Smoker	95% CI
All Vegetables	77.5	69.4,85.6	79.6	72.2,87.0	77.8	72.2,83.4	70.6	63.1,78.1	80.2	74.2,86.1	70.6	62.5,78.6
Garden	43.8	34.3,53.3	42.1	33.3,50.9	52.5	41.8,63.3	41.2	33.1,49.3	58.6	50.1,67.1	43.6	34.9,52.4
Deep Yellow	6.3	2.6,9.9	6.4	2.4,10.4	8.7	2.3,15.0	7.4	4.0,10.8	17.8	12.8,22.8	6.5	3.0,10.1
Cruciferous	21.1	12.5,29.7	18.9	12.0,25.9	23.7	13.4,33.9	21.3	15.1,27.5	28.4	20.7,36.2	22.6	11.8,33.4
Starchy Fibrous	22.5	18.3,26.7	18.3	11.4,25.3	17.6	12.7,22.5	13.2	8.5,17.9	18.8	11.0,26.5	27.0	17.8,36.2
Salad	34.5	27.7,41.3	25.7	18.7,32.6	26.7	19.8,33.7	26.4	21.5,31.4	25.1	17.7,32.5	17.1	9.6,24.6
All Fruits	62.0	54.4,69.5	47.8	38.0,57.5	61.2	53.4,69.0	49.2	40.6,57.8	64.0	56.0,72.0	45.3	37.2,53.5
Fruits/Vegetables												
High in Vitamin A	21.6	13.6,29.7	19.3	12.4,26.2	26.2	16.1,36.2	25.0	17.6,32.4	38.6	30.9,46.3	22.7	12.2,33.2
High in Vitamin C	34.7	26.9,42.6	30.7	23.8,37.6	33.5	25.3,41.6	29.7	23.9,35.5	44.7	35.3,54.0	32.7	22.6,42.8
High Fiber Grains	8.9	5.0,12.8	4.9	1.6,8.2	11.6	6.5,16.6	6.4	3.0,9.8	10.9	6.9,14.8	5.8	0.0*,11.9
Red Meat	58.2	49.6,66.8	56.3	49.7,62.8	44.8	38.8,50.8	45.9	38.4,53.4	35.6	28.7,42.4	35.1	27.2,42.9
Poultry/Fish	47.4	36.5,58.4	41.2	31.7,50.7	55.3	48.5,62.1	48.2	40.3,56.2	52.2	44.4,60.1	47.8	36.0,59.5
Bacon/Luncheon Meat	40.4	34.5,46.2	47.8	37.1,58.6	41.8	35.6,48.0	48.2	41.3,55.2	45.7	39.6,51.9	52.8	43.8,61.8
Whole Milk	41.4	34.8,48.1	35.0	28.2,41.7	27.1	18.0,36.2	32.9	24.6,41.2	33.8	24.7,42.9	33.6	24.4,42.8
Low-Fat Milk	3.5	0.3,6.8	2.7	0.3,5.2	18.7	11.9,25.5	4.5	1.1,7.9	15.7	9.1,22.3	10.2	4.3,16.0
Use Supplements[b]	13.3	8.9,17.7	13.6	7.9,19.2	23.8	16.3,31.3	12.4	8.5,16.3	2.0	14.5,25.5	16.6	9.1,24.0

a) CI = Confidence interval.
b) Data based upon reported "regular" usage of vitamin/mineral supplements.
*Lower limit truncated to 0.0 from a negative value.

Univariate analyses (Tables 1 and 2) showed that non-smokers compared to smokers tended to consume equal amounts of red meat, but more poultry/fish and skim milk, and less bacon/luncheon meats, and whole milk. These findings were confirmed in the multivariate model. In addition, among Whites and most Blacks, non-smokers were more likely to take vitamin/mineral supplements. Blacks, however, were low supplement users overall.

Nutrient Intake Differences

Tables 3 and 4 present, for Whites and Blacks, respectively, univariate analyses of mean intake data for energy, seven nutrients, dietary fiber, and cholesterol by age, sex, and current smoking status. Analyses were also performed for calcium, iron, thiamin, riboflavin, niacin, oleic acid, and linoleic acid, but results showed few or no differences by smoking status (data available upon request to author). As found in past research of the NHANES II data set,[27] smokers tended to consume more kcal than non-smokers. Few intake differences between smokers and non-smokers were found with respect to fat, protein, carbohydrate, and cholesterol.

Mean vitamin C intake is higher among non-smokers in all age-race-sex categories (Tables 3 and 4) with the difference being most striking among White males. After controlling for age, kcal, PIR, and sex, a negative association remained between smoking and vitamin C consumption for both races.

Univariate analyses of vitamin A intake are inconsistent among age-sex-race categories with non-smokers consuming greater or equal amounts compared to smokers. In multivariate analysis, however, a negative association was found between smoking and vitamin A intake among Whites.

With the exception of older Black males, there is a tendency among all age-race-sex groups for smokers to consume less folate and dietary fiber than non-smokers (Tables 3 and 4). After adjustment, these results were confirmed for dietary fiber in both Blacks and Whites and for folate in Whites only.

Linear Trends in Nutrient and Food Intake

Using multiple linear regression, nutrient intake was examined for a linear relationship between sex-specific quintile of nutrient intake and cigarette consumption, as measured in half packs of cigarettes consumed. No trends were found among Blacks for any food or nutrients after adjusting for age. In Whites, after adjusting for age, negative associations between cigarette consumption and both vitamins C and A were found for males ($\beta = -0.11$ s.e. = 0.015, $\beta = -0.06$ s.e. = 0.012, respectively) and females ($\beta = -0.14$ s.e. = 0.014, $\beta = -0.08$ s.e. = 0.014, respectively). Similar negative associations were found for folate among males ($\beta = -0.06$ s.e. = 0.014) and females ($\beta = -0.09$ s.e. = 0.017) and for dietary fiber among males ($\beta = -0.09$ s.e. = 0.015) and females ($\beta = -0.11$ s.e. = 0.017). A positive association was found between cigarette consumption and intake of saturated fatty acids for males ($\beta = 0.04$ s.e. = 0.013).

For food group intake adjusted for age, a negative linear trend existed between increasing cigarette consumption and intake of any vegetable (males $\beta = -0.10$ s.e. = 0.028 and females $\beta = -0.10$ s.e. = 0.029), garden vegetables (males $\beta = -0.09$ s.e. = 0.020 and females $\beta = -0.10$ s.e. = 0.027), any fruit (males $\beta = -0.22$ s.e. = 0.027 and females $\beta = -0.34$ s.e. = 0.030), fruits or vegetables high in vitamin A (males $\beta = -0.13$ s.e. = 0.035 and females $\beta = -0.18$ s.e. = 0.045), fruits or vegetables high in vitamin C (males $\beta = -0.21$ s.e. = 0.031 and females $\beta = -0.19$ s.e. = 0.035), high fiber grains (males $\beta = -0.23$ s.e. = 0.038 and females $\beta = -0.15$ s.e. = 0.048), low fat milk (males $\beta = -0.17$ s.e. = 0.025 and females $\beta = -0.22$ s.e. = 0.040), and vitamin and mineral supplements among all Whites. In White males a negative association was found between increasing consumption of cigarettes and salad ($\beta = -0.08$ s.e. = 0.018), any cruciferous vegetable ($\beta = -0.09$ s.e. 0.032), and poultry and fish ($\beta = -0.07$ s.e. = 0.023).

Discussion

Several limitations of this study should be mentioned. Twenty-four recalls are considered suitable for the study of

TABLE 3—Weighted Mean Nutrient Intake for White Subjects from NHANES II 24-Hour Recall Data by Sex and Smoking Status

	White Men				White Women			
Nutrient	Non-Smoker	95% CI[a]	Smoker	95% CI	Non-Smoker	95% CI	Smoker	95% CI
Ages 19–29 years								
Energy kcal	2947	2790,3105	2997	2868,3127	1667	1614,1720	1679	1603,1755
Protein g	118	112,125	112	107,118	66	64,68	61	57,66
Carbohydrate g	315	300,330	313	297,330	193	186,200	191	182,201
Fat g	122	114,129	118	111,125	68	66,71	66	63,70
Saturated fat g	44	41,47	44	42,47	24	23,25	24	22,25
Cholesterol mg	463	422,505	448	417,479	268	252,284	251	226,275
Vitamin A IU	6000	5368,6631	4529	4155,4903	4582	4156,5007	3810	3142,4477
Vitamin C mg	135	120,150	97	86,108	96	88,104	86	74,98
Folate µg	349	326,372	307	285,330	212	201,223	182	167,198
Fiber g	14.7	13.6,15.7	12.4	11.4,13.4	9.8	9.1,10.5	8.2	7.6,8.8
Ages 30–54 years								
Energy kcal	2444	2340,2548	2569	2493,2645	1535	1488,1582	1591	1542,1641
Protein g	97	93,101	99	96,102	62	60,65	60	58,63
Carbohydrate g	261	248,275	261	254,269	173	168,178	169	163,175
Fat g	102	98,107	108	104,112	63	61,66	68	65,70
Saturated fat g	37	35,39	40	38,41	22	21,23	24	23,25
Cholesterol mg	441	412,469	453	424,482	284	265,302	297	276,318
Vitamin A IU	5796	5112,6480	5926	4854,6997	5567	4726,6409	4204	3676,4732
Vitamin C mg	110	103,117	88	80,95	103	95,111	70	63,76
Folate µg	287	270,305	274	256,292	216	203,229	184	171,198
Fiber g	13.9	13.0,14.7	12.2	11.3,13.1	10.2	9.7,10.6	8.2	7.8,8.6
Ages 55–74 years								
Energy kcal	1998	1955,2042	2010	1942,2078	1360	1329,1391	1390	1336,1445
Protein g	81	79,83	78	75,82	54	52,55	54	51,57
Carbohydrate g	218	212,224	208	199,217	163	158,167	154	146,161
Fat g	82	79,85	85	81,89	54	52,55	57	54,60
Saturated fat g	29	28,30	31	30,33	18	18,19	20	19,21
Cholesterol mg	396	374,418	433	410,456	252	242,262	255	239,272
Vitamin A IU	6320	5824,6816	5990	5278,6702	5426	5000,5851	5225	4575,5874
Vitamin C mg	111	104,118	88	79,97	109	103,115	93	81,105
Folate µg	281	269,293	255	237,272	217	209,224	200	187,213
Fiber g	13.1	12.5,13.7	11.9	11.0,12.9	10.8	10.6,11.1	9.1	8.5,9.8

a) CI = Confidence interval.

group but not individual means.[46] At least one investigator, however, has suggested that the bias due to reporting error inherent in the method may be greater than the difference detected between groups.[47] Given the large size and scope of NHANES II, the 24-hour recall data are likely to be appropriate for the group analysis presented here, but should be viewed with caution because of potential bias. In particular, conclusions regarding nutrients known to have large variability (such as vitamin A) may be subject to question when small groups are examined.[47] Because the number of Blacks sampled in NHANES II is relatively small in comparison to Whites, the results for Blacks cannot be viewed with the same degree of confidence. Finally, the coding of intake of food groups which is based upon mentions from the recalls yields no information regarding portion size and, therefore, is useful but imprecise quantitatively.

Smoking and diet are among the top 10 risk factors thought to cause the majority of disease in the US.[48] Our findings suggest that there are important dietary differences between smokers and non-smokers, all tending toward unhealthy patterns. Surprisingly few differences by current smoking status were found for fat, saturated fat, and cholesterol, dietary variables associated with coronary heart disease. Smokers did, however, consume less poultry/fish and low fat milk and more whole milk and luncheon meats than non-smokers. With respect to cancer prevention, however, there is interest not only in the role of fat, but also in the roles of fruits, vegetables, fiber, vitamin A/carotenoids, vitamin C, and folate. For all these foods or dietary constituents, we found that smokers, in general, consume less than non-smokers, with vitamin C/fruit intake showing the greatest and most consistent difference across all age-sex-race groups.

Our findings with respect to vitamin C and fruits are intriguing for several reasons. First, vitamin C is thought to protect against cancer through reduction functions which serve to both prevent carcinogens from forming and decrease the carcinogenic effects of certain chemical agents and through the enhancement of host resistance.[49] Second, several studies have shown that smokers have lower serum vitamin C levels than non-smokers, and that vitamin C turnover in smokers is higher than in non-smokers.[31,50–52] Several investigators have suggested, therefore, that smokers have increased vitamin C requirements. While mean vitamin C intake data suggest that smokers are meeting their Recommended Dietary Allowance (RDA) for vitamin C (60 mg),[53] this level may not be high enough to counteract the increased vitamin C turnover resulting from their smoking habit. Our data suggest that this very group, which requires more vitamin C, is consuming significantly less. While we do not account for vitamin C intake from supplements, the data show that smokers are less likely to be taking supplements than non-smokers. Third, our vitamin C and fruit results are

TABLE 4—Weighted Mean Nutrient Intake for Black Subjects from NHANES II 24-Hour Recall Data by Sex and Smoking Status

	Black Men				Black Women			
Nutrient	Non-Smoker	95% CI[a]	Smoker	95% CI	Non-Smoker	95% CI	Smoker	95% CI
Ages 19–29 years								
Energy kcal	2603	2247,2958	2796	2348,3244	1715	1576,1855	1731	1573,1889
Protein g	102	88,116	110	76,145	67	62,72	61	55,67
Carbohydrate g	281	241,320	281	253,308	202	176,227	211	187,235
Fat g	110	91,129	114	87,140	71	67,76	67	61,74
Saturated fat g	38	31,44	42	30,54	25	23,28	23	20,26
Cholesterol mg	487	396,578	547	360,735	321	269,372	341	298,383
Vitamin A IU	7973	2410,13536	6530	4701,8358	3923	3306,4539	3987	2829,5145
Vitamin C mg	129	98,160	113	89,138	108	79,136	107	84,130
Folate μg	302	248,356	247	219,274	194	160,227	188	156,220
Fiber g	12.8	10.4,15.2	10.2	8.9,11.5	8.3	7.3,9.3	7.7	6.4,9.0
Ages 30–54 years								
Energy kcal	2006	1784,2228	2369	2140,2598	1391	1268,1514	1328	1239,1418
Protein g	81	70,92	88	81,95	55	50,59	52	47,58
Carbohydrate g	223	204,242	229	209,249	159	143,176	145	129,160
Fat g	82	70,94	93	80,106	57	51,63	56	49,62
Saturated fat g	28	24,33	33	28,39	20	17,22	19	17,21
Cholesterol mg	514	373,655	472	411,533	291	252,331	305	262,349
Vitamin A IU	10204[*]	6004,14405	4943	3225,6661	4503	3528,5478	6008	4498,7518
Vitamin C mg	133	89,177	104	74,133	96	76,116	71	59,82
Folate μg	264	208,319	213	195,232	173	153,193	155	140,170
Fiber g	12.7	10.3,15.1	9.3	8.4,10.4	7.5	6.8,8.2	7.2	5.9,8.5
Ages 55–74 years								
Energy kcal	1751	1579,1924	1764	1617,1912	1255	1167,1343	1341	1158,1524
Protein g	69	62,76	68	62,75	51	47,54	55	45,64
Carbohydrate g	191	168,214	177	161,193	150	138,162	158	141,176
Fat g	71	63,80	73	64,83	50	45,55	53	45,64
Saturated fat g	25	22,29	27	23,30	17	15,19	18	15,22
Cholesterol mg	378	342,415	420	363,477	273	231,315	261	206,317
Vitamin A IU	6727	4632,8821	5591	4048,7134	7667	5592,9741	4718	2430,7006
Vitamin C mg	98	76,121	79	62,95	114	100,129	105	61,150
Folate μg	216	186,246	241	190,292	190	156,223	175	143,208
Fiber g	11.8	9.8,13.9	10.1	7.8,12.5	9.0	7.9,10.1	9.4	7.9,10.9

a) CI = Confidence interval.

*High value due to 3 individuals with unusually large intakes of liver and/or eggs and/or carrots.

consistent with research suggesting that smoking (via nicotine) decreases the desire for sweet foods and in particular, fruit juices.[54,55] While our results may, in part, be explained by taste preference characteristics due to the metabolic effects of nicotine, this does not resolve the differences found between smokers and non-smokers in their intake of other non-sweet foods.

High intakes of dietary fiber are suspected to be a preventive factor in colon cancer,[56] and the National Cancer Institute recommends that individuals consume 20–30 gm of dietary fiber daily.[57] Past research based on NHANES II data shows that intake of dietary fiber in the US is much lower than this recommendation,[37] and we show that smokers have among the lowest intakes. While tobacco use is not known to be related to bowel cancer, our results suggest that smokers could be at higher risk for this cancer through related dietary lifestyle habits. Furthermore, our results indicate that this lower intake results from lower consumption of several key sources[58] of dietary fiber: vegetables, whole grains, and fruits.

Consistent with past research,[30,32] we found that smokers have, for the most part, lower intakes of vitamin A than non-smokers. While smokers have 25 times the risk of developing lung cancer compared to non-smokers, both low dietary intake and serum vitamin A and/or carotenoids have been shown to increase smoking adjusted lung cancer risk in the range of 1.3 to 2.7 times.[13,14,16,19–25] Furthermore, several studies have shown that low dietary or serum vitamin A/carotenoid levels increase lung cancer risk (range: 1.3–8.1) among smokers, including the heaviest and those who have smoked the longest.[12,16,18,20,22,24,25] Evidence of dietary protection is strongest for carotenoids (from plants) versus retinol sources (from animals).[59] A separate measure of the carotenoid or retinol composition of foods was not available in our database, but our finding that smokers consume fewer fruits or vegetables high in vitamin A, and fewer fruits and vegetables overall, suggest that smokers have lower carotenoid intakes than non-smokers. Stryker, *et al*,[28] have suggested that smokers need to increase their intake of carotenoids to achieve blood levels comparable to their non-smoking counterparts. Our results show that smokers, who may gain some protection from lung cancer by high carotenoid intake, are less likely than non-smokers to be consuming carotenoid-dense foods. Furthermore, former smokers, who enjoy gradually declining lung cancer risk, should be encouraged toward improved nutrition in an effort to possibly accelerate the decline.

A few reports have suggested that folate may be protective for both lung and cervical cancer.[60–62] While our results with respect to intake differences between smokers and non-smokers are less striking for folate than for vitamin C, such differences should be noted in that they fit into a general pattern of lower intake of fruits, vegetables and high fiber grains among smokers.

Questions remain as to whether specific dietary factors such as vitamin A, carotenoids, and vitamin C are the operative cancer protective factors in foods or whether some other yet unknown factor(s) is responsible. Studies have shown that intake of cruciferous vegetables, fruits and vegetables, and especially yellow-orange and green vegetables have protective effects not only for lung cancer, but also for breast, colon, prostate, bladder, oral cavity, stomach and cervical cancers.[5,6] Our results show that intake of all these foods is significantly lower among smokers. This suggests that smokers may be at higher risk for many cancers, some of which are non-smoking-related. Of particular interest is our finding that for several of the cancer protective food groups, there are modest but consistent trends for intake to decrease as cigarette consumption increases. Thus, the more one tends to smoke, the more one increases cancer risk, the less one tends to consume fruits, vegetables, and high fiber grains. These findings are similar to that of Schoenborn and Benson[8] regarding other lifestyle factors such as sleeping less than six hours per night, skipping breakfast, being less physically active, being sedentary, and drinking heavily.

One possible explanation for lower intakes of nutrients and some foods among smokers may be that smokers compared to non-smokers tend to be greater consumers of alcoholic beverages[5] and, therefore, may replace calories and nutrients from food sources with those from drinks. Because we had no data regarding the contribution of alcohol to either the caloric or carbohydrate content of the 24-hour recalls we analyzed, we were not able to explore this possibility, and past research yields conflicting results.[29,63] Further research is necessary to determine whether alcohol consumption plays a significant role in the lower intake of nutrients and foods found among smokers in this research.

Our findings of lower intakes of vitamin A, vitamin C, fiber, and folate among smokers are correlated in that all these nutrients are common to vegetables, fruits, and grains, foods for which we also found lower consumption among smokers. The task of understanding how nutrients and/or foods protect or promote cancer while considering other risk factors such as smoking is difficult. We have shown that smokers, who are known to be at increased risk for several cancers, have lower intake of nutrients and foods associated with cancer prevention than non-smokers. The evidence is clear that smoking is a habit which leads to or is lead by other lifestyle practices, and we have shown that yet another important health habit is associated with smoking: food intake. This suggests that analyses in future epidemiological research regarding cancer prevention or etiology account for the possible confounding of smoking and diet.

Smoking cessation is the single biggest prevention measure for purposes of cancer prevention and control, particularly with respect to lung cancer.[2] Individuals who smoke are clearly at greatly increased risk from their tobacco use. However, this study suggests that they may be at some slightly additional risk from dietary intake low in critical nutrients. While more research is needed regarding smoking and diet, smokers and ex-smokers may be well advised to increase their intake of fruits, vegetables, and high fiber grains. In the meantime, physicians and the public should be aware that some smokers have low intakes of foods or nutrients associated with decreased cancer risk.

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Depressive Mood, the Single-Parent Home, and Adolescent Cigarette Smoking

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Abstract: The association between depressive mood and cigarette smoking among adolescents was examined within a multivariate model. Subjects were 205 eleventh graders (123 boys and 82 girls) enrolled in a Northeast metropolitan public high school for science-oriented students. Logistic regression analysis showed an independent relation of depressive mood, friends' smoking behavior, and living in a single-parent home with cigarette smoking. Depression scores correlated with the number of cigarettes smoked. These associations suggest that depressive mood and stress may contribute to the onset of smoking. (*Am J Public Health* 1990; 80:1330–1333.)

Introduction

A host of factors have been investigated in numerous attempts to determine the antecedents of cigarette smoking among adolescents. Peer and family smoking behavior have been consistently implicated[1-3]; while less evidence has been demonstrated for the effects of psychological traits, knowledge and attitudes about smoking, school achievement, and demographic characteristics.[1]

A prospective study involving 1,004 subjects found that depressive symptoms reported at ages 15–16 predicted the frequency and duration of cigarette smoking nine years later.[4] This association was not seen for future alcohol or drug use. Another study found higher scores on the Beck Depression Inventory among cigarette smokers than in nonsmokers in males.[5] Both studies limited analysis to simple associations; neither controlled for the effect of other known risk factors. We have found no other attempts to examine the relation between depression and smoking among adolescents. In contrast, a recent paper cited seven studies that examined the relation between depressive disorders and drug and alcohol abuse.[6]

The present study reports our investigation into the relation between depressive symptoms and cigarette smoking among adolescents within the context of a multifactorial model.

Methods

Subjects were 205 11th graders (123 boys and 82 girls) enrolled in a public science high school in a Northeast metropolitan area. This sample included all subjects present on the day our questionnaire was administered in seven non-elective health education classes; they comprised 35 percent of the school's total 11th grade population in the school year 1987–88. Enrollment into this school is competitive and requires passing a qualifying examination. We did not obtain ethnic information from our subjects. School records, however, showed the following ethnic breakdown for the total 11th grade class: 45 percent White, 33 percent Asian, 14 percent Black, 8 percent Hispanic, 0.5 percent American Indian. It is unlikely that our sample deviated markedly from the total 11th grade class in ethnic distribution.

To enhance compliance and candid reporting, we developed a questionnaire that was short (limited to a single, double-sided page), anonymous, and noninclusive of potentially sensitive areas (e.g. grade point average, ethnicity, use of alcohol or drugs). Certain items were not answered by some respondents, accounting for the uneven group totals shown in the accompanying tables.

Subjects were asked whether or not they were regular smokers (at least one cigarette a day), the amount they smoked, the age of smoking onset, and the brand of cigarettes most frequently smoked. We also asked about the smoking behavior of parents, siblings, and friends; estimated parents' annual income; and number of siblings.

We selected 15 of the 20 (CES-D) items[7] to measure level of depressive symptomatology. The feasibility of using this scale with adolescents[8] and that of using a shortened version[9] have been demonstrated. To determine the specificity of an effect of depression, we included two other mental health measures: seven items from an inventory of "worries" used with schoolchildren[10] and from other instruments, assembled seven items assessing level of satisfaction with various life concerns (Appendix). Internal reliability (Cronbach) coefficients based on the total sample were 0.81 for the depression scale, 0.74 for worry, and 0.73 for satisfaction items.

We measured family structure by asking if the subject lived with one or both parents but did not determine whether the custodial parent was separated, divorced or widowed.

Yates corrected chi square analyses and t-tests were used in comparisons of boys vs girls, smokers vs nonsmokers, and highly exposed vs less exposed smokers. Multiple logistic regression[11] using a model that included all the study variables was employed to test the independence of observed associations with cigarette smoking. Quantitative measures were dichotomized at their median values.

Results

We found an overall similarity between boys and girls on most of the psychosocial and smoking variables we studied (Table 1). The mean age of the sample was 16.1 (s.d. = 1.7) years. They were predominantly middle class (about 50 percent estimated their family income to be $25,000–$50,000), and living in two-parent households (73.6 percent). Only two children were not living with either parent.

The group prevalence of regular smoking (at least one cigarette daily) was 26.8 percent. It was higher in boys, who also reported smoking more cigarettes, than in girls. Girls reported a lower smoking rate among their mothers than their fathers, a difference that was not seen among the boys (Table 1).

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TABLE 1—Psychosocial and Smoking Characteristics of 205 11th Graders by Sex

Characteristics	Boys N = 123 (%)	Girls N = 82 (%)	Total N = 205 (%)
Age≤16 years	85 (70.2)	57 (69.5)	142 (70.0)
Mean age (s.d.)	16.0 (2.1)	16.3 (0.6)	16.1 (1.7)
Family income			
<$25,000	17 (16.3)	8 (12.1)	25 (14.7)
$25,000–$49,999	49 (47.1)	36 (54.5)	85 (50.0)
$50,000>	38 (36.5)	22 (33.3)	60 (35.3)
Not the only child	96 (78.0)	63 (76.8)	159 (77.6)
Living with both parents	93 (76.2)	57 (69.5)	150 (73.6)
Regular smokers	34 (27.6)	21 (25.6)	55 (26.8)
Frequency of smoking			
1 cigarette/day	14 (41.2)	12 (60.0)	26 (48.1)
About 10–20	10 (29.4)	6 (30.0)	16 (29.6)
More than 20	10 (29.4)	2 (10.0)	12 (22.2)
Began smoking <15 yrs	19 (55.9)	12 (57.1)	31 (56.3)
Mean age began (s.d.)	14.1 (1.4)	15.5 (6.1)	14.7 (4.0)
Smoke brand with ≥1 mg nicotine	26 (76.5)	10 (52.4)	36 (66.3)
Friends who smoke			
Most or All	23 (22.1)	13 (17.6)	36 (20.3)
Some	49 (47.1)	34 (45.9)	83 (46.6)
None	32 (30.9)	27 (36.5)	59 (33.1)
Mother smokes:	26 (27.1)	9 (11.1)	35 (20.7)
Father smokes:	25 (26.6)	21 (25.9)	46 (27.5)
Siblings smoke:	7 (8.4)	8 (9.9)	15 (10.8)

Note: Percents shown are based on group totals that do not include missing answers.

Mean scores for each of the three mental health variables, i.e. depression, worry, and satisfaction, did not differ by sex. Depression was positively correlated with worry, r = .30, (205) $p < .001$ and negatively correlated with satisfaction, r = −.36, (205) $p < .001$. Satisfaction and worry scores were negatively correlated, r = −.16, (205) $p < .02$.

Rates of cigarette smoking by selected factors are shown in Table 2. The most striking association with cigarette smoking was observed for peer smoking behavior. Smokers were also more depressed and worried, and less satisfied, than were nonsmokers (Table 3).

TABLE 2—Prevalence of Regular Smoking by Selected Characteristics in a Sample of 205 11th Graders

Characteristics	% Smokers	Total N
Age		
≤16 years	23.7	142
>16 years	37.7	61
Family income		
<$25,000	28.0	25
$25,000–$49,999	28.2	85
$50,000>	28.3	60
Only child		
Yes	32.6	46
No	25.2	159
Live with both parents		
Yes	22.7	150
No	37.0	54
Father smokes		
Yes	39.1	46
No	29.8	121
Mother smokes		
Yes	45.7	35
No	29.1	134
Siblings smoke		
Yes	53.3	15
No	27.6	123
Friends smoke		
All	100.0	6
Most	80.0	30
Some	24.1	83
None	8.5	59

Note: Missing answers account for uneven group totals.

Multiple regression analysis (Table 4) showed that three variables were independently related with smoking status. As in the univariate analyses, the strongest relationship was observed for friends' smoking behavior (odds ratio = 7.7). An excess risk for smoking was also seen for respondents who scored above the group median (11>) on the shortened CES-D scale (odds ratio = 3.9), and for those who lived in a single-parent home (odds ratio = 1.9). Only children showed some risk for being a smoker but the confidence interval of the odds ratio (1.6) included 1. No interaction term was associated with smoking.

Smokers who smoked a pack or more a day reported a depression score of 22.7 compared to 15.4 for smokers of fewer cigarettes (difference = 7.3; 95% CL = 2.5, 12.1). No relation between age began smoking or nicotine yield of brand smoked and depressive symptoms was observed.

Discussion

Our sample was drawn from a metropolitan high school for talented, high-achieving students. We can only speculate about the extent to which the competitive and demanding climate of their school environment engendered the preva-

TABLE 3—Mean (standard deviation) Scores of Regular Cigarette Smokers (n = 150) and Nonsmokers (n = 55) in a Sample of 205 11th Graders on Scales Measuring Depression, Worry, and Satisfaction

	Nonsmokers		Smokers		Difference (95% CI)
	Mean	(SD)	Mean	(SD)	
Depression	10.7	(6.4)	17.5	(7.9)	6.8 (4.7, 8.9)
Worry	8.9	(2.5)	9.8	(2.9)	0.9 (0.1, 1.7)
Satisfaction	28.3	(6.7)	25.7	(7.3)	2.6 (0.5, 4.7)

TABLE 4—Multiple Logistic Regression of Smoking Status, Nonsmokers (0) vs Smokers (1) on Selected Characteristics of 205 11th Graders

Independent Variables	Adjusted Odds Ratio	95% CI
Friends smoke (0 = None, Some; 1 = Most, All)	7.70	3.15, 18.8
Depression (0 =<11; 1 = 11>)	3.86	2.56, 5.82
Family structure (0 = 2 parents; 1 = 1 parent)	1.92	1.01, 3.71
Only child (0 = No; 1 = Yes)	1.62	.53, 4.95
Siblings smoke (0 = No; 1 = Yes)	1.13	.50, 2.59
Family income (0 = <$25,000; 1 = $25,000>)	.91	.48, 1.73
Age (0 = <16; 1 = 16>)	.90	.48, 1.68
Father smokes (0 = No; 1 = Yes)	.91	.44, 1.86
Mother smokes (0 = No; 1 = Yes)	.72	.33, 1.57
Worry (0 = <9; 1 = 9>)	.68	.37, 1.25
Satisfaction (0 = <28; 1 = 28>)	.57	.29, 1.16

Likelihood ratio $\chi^2 = 41.84$, df = 11, $p < .001$

lence of depressive symptoms in our sample or the higher rate of smoking we observed (27 percent) compared to the rate seen in similar aged national samples (18 percent).[12] While these special characteristics of our subjects may limit the generalizability of our findings, they do not reduce their validity.

The strong and independent relationship between depressive symptoms and cigarette smoking confirms previous results obtained prospectively[4] and in a cross-sectional design.[8] The correlational nature of our study does not permit us to infer a causal relation or the direction of a causal sequence involving cigarettes and depression. However, neither a direct nor indirect effect from cigarette smoking to depression is likely. There is no evidence that nicotine, the major pharmacological agent in tobacco, causes depression,[13] and our data did not show more depression among smokers of high nicotine-content tobacco. Also, we did not observe that having more friends who smoked (which may reflect membership in a minority, deviant group) is correlated with higher depression. On the other hand, Kandel's observation[4] based on longitudinal data that cigarette smoking in adulthood is a sequela of depressive symptoms in adolescence supports an antecedent, if not causal, relation of depressive affect with cigarette smoking. An association between clinical depression and smoking status has been found in adult samples.[14,15] It remains to be seen whether such an association exists among adolescents as well since the CES-D, while potentially useful as a screening instrument, does not diagnose clinical depression.[16]

The rate of smoking among subjects living in single-parent homes was higher than the smoking rate in other subjects (37 vs 22 percent, respectively), an association that was independent of peer smoking behavior or depression rating. This finding agrees with an earlier observation that disruptive family events are directly associated with adolescent cigarette smoking.[17] In accord with Wills'[18] coping model of smoking in adolescence, cigarette smoking may be an attempt to mitigate the stresses brought on by parents' marital break-up. This explanation is compatible with the notion that parents' divorce may exacerbate the trend towards maladaptive coping among adolescents.[19] We offer these speculations with caution, however, since we obtained only limited information regarding the circumstances surrounding parents' separation.

Our results suggest that merely teaching youngsters how to "just say no" to their peers may not be enough. Depressive mood and parental separation appear to be additional risk factors contributing to the prevalence of smoking among adolescents. Multicomponent prevention approaches that will also address adolescents' emotional needs and the strains engendered by the home situation are indicated.

APPENDIX

Depression Items (15 from the CES-D Scale)

Below is a list of the ways you might have felt or behaved. Please indicate your answer.

a) Rarely or none of the time (less than 1 day)
b) Some or a little of the time (1–2 days)
c) Occasionally or a moderate amount of time (3–4 days)
d) Most or all of the time (5–7 days)

During the past week:

A. I was bothered by things that usually don't bother me
B. I did not feel like eating; my appetite was poor.
C. I felt that I could not shake off the blues.
D. I felt that I was just as good as other people.
E. I felt depressed.
F. I felt hopeful about the future.
G. I thought my life had been a failure.
H. My sleep was restless.
I. I was happy.
J. I talked less than usual.
K. I felt lonely.
L. People were unfriendly.
M. I had crying spells.
N. I felt that people dislike me.
O. I could not get "going".

Worry Items

Almost everyone at one time or another is worried about something. Different people worry about different things. Read and check appropriately if it worries you.

	A lot	A little	Hardly ever
1. Not getting along with your parents	—	—	—
2. Not being popular	—	—	—
3. Feeling left out of things	—	—	—
4. Being made fun of	—	—	—
5. Being looked down upon	—	—	—

Satisfaction Items

For each area of life, choose the letter that corresponds with the level of satisfaction you get from that area.

A) a very great deal
B) a great deal
C) quite a lot
D) a fair amount
E) a little
F) none

a) city or place you live in ____
b) school work ____
c) hobbies or sports activities ____
d) extracurricular activities ____
e) family life ____
f) your friendships ____
g) your physical condition ____

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Effects of Individually Motivating Smoking Cessation in Male Blue Collar Workers

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Abstract: Adverse demonstrable health effects linked to the individual's smoking were shown to 2,689 American workers to motivate cessation during routine examinations to detect asbestosis. This intervention was evaluated six to 25 months later by a mailed questionnaire and by telephone to non-responders. Results were compared to yearly quit rates of 2.5 percent to 5 percent for 736 workers who were ex-smokers at the initial examination. Of the 504 men who responded by mail, 29.8 percent had quit smoking, 35.9 percent had cut down from a mean of 28 to 13 cigarettes per day, and 34.3 percent were smoking as before. Subsequent follow-up at one year showed that 25.6 percent remained quit, and that 23 percent of those who cut down had quit, for an overall quit rate of 34 percent. Of 101 non-responders contacted by telephone, 17 percent had quit and 53 percent had reduced smoking. In both samples, those who quit were more likely to have had lower alveolar carbon monoxide (CO_a) levels, to be older, and to have had asbestosis. Responders by mail were the same age as non-responders but had smoked longer, had higher prevalences of asbestosis, emphysema, chronic bronchitis and higher CO_a. Demonstration of the adverse personal effects of smoking appear to have contributed to the quit rates or reduced smoking rates in 65 percent of the responding workers. (*Am J Public Health* 1990; 80:1334–1337.)

Introduction

Smoking cigarettes is the largest preventable cause of illness and premature death in the United States. It is responsible for 30 percent of all cancer deaths.[1] Asbestos is the single most important occupational cause of lung cancer.[2] Occupational asbestos exposure raises the lung cancer risk five times and combined with cigarette smoking increases the risks 50- to 100-fold.[3] Smoking and asbestos are synergistic in producing pulmonary fibrosis and functional impairment as well.[4] Despite the fact that blue collar workers in trades exposed to asbestos usually smoke more than others in the work force,[4] few efforts have been aimed at modifying their smoking.[5] In one reported exception, 65 percent of smokers in a California shipyard had quit smoking, leaving only 18 percent of current workers still smoking.[6] This was attributed to their being made aware of their own health risks of smoking combined with asbestos exposure. Thus, we wondered whether other asbestos exposed workers could be motivated to quit smoking if a physician and other health workers pointed out the adverse effects of smoking on their own body and bodily functions. We compared the effects of this intervention to the yearly quit smoking rates in workers who were ex-smokers at the time of study.

Methods

The men studied had been exposed to asbestos in construction trades and shipbuilding as active or retired members of the International Brotherhood of Boilermakers, Iron Ship Builders, Blacksmiths and Forgers, the United Association of Plumbers and Pipefitters or other unions. All men were White . They had to have been exposed to asbestos at least 15 years earlier and for a duration of at least five years. Ages ranged from mid-thirties to mid-eighties. We screened 4,183 of these men from 20 locations in the United States, from Oregon and Southern California to Florida and North Dakota. There were 19.6 percent who had never smoked, 17.6 percent were ex-smokers, and 62.8 percent were current smokers. The intervention target was 2,627 current smokers who received our follow-up letter. The comparison populations for smoking cessation rates were 134 men who had quit in the preceding year and 736 men classified as ex-smokers by interview backed by alveolar carbon monoxide levels.

All men had completed questionnaires in their union halls concerning respiratory symptoms, onset, intensity and duration of cigarette smoking, occupational exposures and exercise tolerance. They had been given a brief physical examination including measurement of height, weight, pulse rate, blood pressure, auscultation of the chest and presence of clubbing, cyanosis, and edema. Chronic bronchitis was diagnosed from the criteria of cough and sputum production for more than three months of two consecutive years.

Smoking status reported on questionnaire, was verified by measuring the level of carbon monoxide in alveolar gas (CO_a) during 20 seconds of breath holding with a fuel cell.[4] Vital capacity, forced expiratory volume in one second and flow rates,[7] were measured by spirometry. Each worker's measurements were expressed as percentage of predicted obtained by regression equations.[8] Total lung capacity was measured by planimetering of posteroanterior and lateral chest x-rays, obtained at full inflation of the lungs for area and volumes calculated.[9]

To motivate quitting, the effects of cigarette smoking including symptoms (particularly cough and sputum), chest x-ray changes, abnormal pulmonary function tests and findings on physical examination were explained by the team of examiners. The physician made clear the contribution of cigarette smoking when signs of asbestosis were found in the lung or pleura on the worker's x-ray[10] and when signs of emphysema, such as hyperlucent lung fields, hyperinflation, bullae and other abnormalities[11] were present. He also related these findings to shortness of breath and limited endurance in climbing hills and stairs and observed that these symptoms and abnormalities would progress if the worker continued to smoke but would stop progressing if he stopped smoking. The extremely high risk of dying of lung cancer in asbestos-exposed workers who smoke was also emphasized. Elevated alveolar carbon monoxide (CO_a) levels and reduced spirometric flow curves were linked to smoking as were current or eventual shortness of breath by senior technicians.

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During the physical examination a nurse practitioner pointed out to those with diminished breath sounds and wheezes that these were characteristic effects of smoking, which indicated obstruction to airflow which in turn causes shortness of breath. Finally, while assisting workers with occupational, medical and pulmonary histories, interviewers emphasized adverse health effects of smoking including the risks for cancer and impairment of breathing which are enhanced by asbestos exposure.

After data were assembled, a personal letter summarizing the findings was sent to each worker from the physician which enclosed results of his pulmonary function tests with the abnormalities identified. Each letter emphasized the greatly increased risk of lung cancer for asbestos-exposed workers who smoke cigarettes.

From six to 25 months after the asbestos screening and study, a letter was sent to each man who was a current smoker. A brief questionnaire was enclosed querying the worker's recall of the screening and report letter, and his current smoking status (quit, cut down, or continued at the same level). He was asked to report the current number of cigarettes smoked per day and whether he still desired to quit. Decreased smoking was defined by the number of cigarettes per day on re-survey being less than the number reported at the examination.

Letters and questionnaires were mailed to 2,689 current smokers; 50 were returned as not reaching the subject and 12 men responded that they had already quit smoking at the time of the screening, which reduced the base numbers to 2,627.

In the early evening, interviewers questioned by telephone 101 of the non-responders selected at random from the 15 largest study sites across the country. Eleven workers who were not reached by calls placed three times on different evenings were replaced. Interviewers asked the same questions as those in the mailed questionnai[illegible] 101 men recalled the asbestos disease screening [illegible] their being strongly advised to quit smoking. Their responses are compared separately from mail responders.

Fifty of the mail responders selected at random from the 150 who reported quitting and 61 selected at random from the 181 who reported cutting down were asked the same questions in a telephone interview 12 months after the return of the questionnaire. Interviewers did not mention the previous mail questionnaire but, if asked, replied that they were doing an interval follow-up. Data from the mail and telephone questionnaires were merged with the data from the asbestosis and lung disease study[4] and tables were constructed showing means and standard deviations. Stepwise linear regressions were made and a correlation matrix was examined.

Results

The data are based on 504 returned questionnaires, a response rate of 19.2 percent to the mailing, and on the 101 non-responders interviewed by telephone. The mail responders were more likely than the 2,123 non-responders to have smoked longer and to have had asbestosis, emphysema, chronic bronchitis and an elevated CO$_a$ level (Table 1), but were otherwise generally similar to responders. Lapse of time since the original study (six to 25 months) was not associated with differences in response rates or in smoking behavior. The 101 non-responders interviewed by telephone were similar to the mail responders in age (mean of 52 years), mean CO$_a$ (34.8 ppm), and prevalence of asbestosis (33.6 percent), emphysema (15.1 percent), and chronic bronchitis (35.9 percent).

TABLE 1—Comparison of Cigarette Consumption, Occupational Exposure, Pulmonary Disease Prevalence and Mean Pulmonary Function Measurements as Percentage of Predicted (pop) in Men who Responded and Did Not Respond to the Smoking Status Questionnaire

	Responded	Did not Respond
Number	504	2123
	mean sd	mean sd
Age yrs	52.9 ± 10.9	53.7 ± 12.0
CO$_a$ ppm	33.0 ± 17.0	22.1 ± 18.3
Duration Smoking yrs	34.2 ± 12.3	27.0 ± 13.4
Number Cig/day	28.3 ± 14.8	28.2 ± 14.8
Cigarette consumed pack yrs	47.9 ± 30.6	3.97 ± 29.6
Current Cig/day	20.4	—
Duration Welding yrs	15.4	14.7
Employed in Shipyards, as %	48.3	48.5
Duration Asbestos Exp. yrs	26.8 ± 10.5	27.7 ± 11.1
Chronic Bronchitis %	38.1	27.7
Asbestosis pleural %	17.1	17.8
Asbestosis parench. %	38.3	28.0
Asbestosis diagnosed, total %	44.0	35.7
Emphysema by x-ray preval. %	15.5	8.3
Bullae prevalence	9.1	4.7
FVC pop	94.7 ± 16.4	94.6 ± 15.8
FEV$_1$ pop	89.5 ± 22.5	90.7 ± 20.2
FEF$_{25-75}$ pop	77.1 ± 42.4	81.4 ± 40.5
FEF$_{75-85}$ pop	75.3 ± 48.9	80.4 ± 46.8
TGV pop	114.2 ± 14.3	110.8 ± 13.6
RV/TGV	45.4 ± 11.4	44.0 ± 11.5
FEV$_1$/FVC	69.0 ± 12.8	71.5 ± 11.6

Significant differences compared to "did not respond" are underlined.

Because we lacked a comparison group, we used as historical controls the reported yearly quit rates for the 736 ex-smokers who had been studied for asbestosis and lung diseases at the same time and under identical circumstances to the cigarette smokers and 134 men who quit smoking during the year preceding the study. These were considered to provide the natural smoking attrition rates, before our intervention. The quit rate reported for the year prior to intervention was 4.7 percent and rates varied from 2.5 percent to 5.5 percent, going back 20 years, to 1969.

Of the mail responders, 29.8 percent (150 of 504) had quit smoking after the intervention or after receiving the letter reminding them of their health status (Table 2). Another 8 (1.6 percent) had quit but resumed by the time of inquiry and thus are recidivist smokers. In addition, 35.9 percent reported they had cut down smoking from a mean of 28 to 13 cigarettes per day. The remaining 32.7 percent reported smoking as before but over 90 percent of them said they were still trying to quit. Thus 65.7 percent of the workers who responded by mail had ceased or reduced smoking after the screening and intervention. In the interval telephone follow-up a year after receipt of the questionnaire, the 50 men who had been classified as quitters by mail said they had quit at that time and 43 (86 percent) had remained off cigarettes; seven (14 percent) were recidivists. If these proportions are applied to the whole group, the 25.6 percent of the mail response quitters were not smoking one year after responding to the mail questionnaire. The one-year telephone interview follow-up of 61 men who indicated in the mail questionnaire they had reduced smoking showed that an additional 14 (23 percent) had quit between inquiries and had remained off cigarettes. Applying these quit rates to the entire 504 responders would

TABLE 2—Comparison of Cigarette Smoking, Occupational Exposure, Pulmonary Diagnoses and Pulmonary Function Status of 504 Responders by Mail by Cigarette Smoking Status

	Smoking Status		
	Quit	Decreased	Unchanged
Number	150	181	165
	mean sd	mean sd	mean sd
Age	53.6 ± 10.9	55.6 ± 11.2	49.0 ± 10.1
CO_a ppm	27.5 ± 16.1	33.9 ± 17.5	36.5
Duration Smoking yrs	34.7 ± 11.8	36.4 ± 13.1	31.6 ± 10.5
Number Cig/day	25.3 ± 14.7	28.5 ± 14.0	30.0 ± 15.4
Pack years	45.6 ± 32.6	51.0 ± 30.8	46.3 ± 28.5
Current Cig/day	0	13.3 ± 8.4	28.8 ± 15.9
Duration Welding yrs	15.5	14.9	15.9
Employed in shipyard, %	53.1	49.0	42.5
Duration Asbestos Exp. yrs	27.5 ± 9.5	27.5 ± 11.7	25.2 ± 9.9
Chronic Bronchitis Hist %	33.1	37.0	44.2
Ashthma History %	17.9	19.3	18.8
Asbestosis pleural %	22.7	22.1	6.0
Asbestosis parenc. %	49.3	47.0	18.0
Bastestosis diag tot. %	54.7	55.2	21.2
Emphysema by x-ray prev %	18.0	13.8	15.2
Bullae preval.	8.0	7.1	13.9
FVC pop	95.6 ± 21.2	92.2 ± 17.1	96.4 ± 16.5
FEV_1 pop	90.1 ± 21.2	87.2 ± 23.8	91.2 ± 22.2
FEF_{25-75} pop	75.7 ± 42.9	73.8 ± 43.5	81.3 ± 40.6
FEF_{75-85} pop	77.0 ± 58.5	72.0 ± 44.9	77.2 ± 43.6
TGV pop	113.8 ± 14.4	114.9 ± 13.7	115.0 ± 14.3
RV/TGV	44.8 ± 11.4	48.2 ± 11.0	43.9 ± 10.9
FEV_1/FVC	68.9 ± 12.5	67.9 ± 13.3	70.4 ± 12.1

Significant differences compared to "unchanged" are underlined.

predict that 171 (34 percent) had quit at 18 to 37 months after the intervention. Those who continued to smoke averaged 15.8 cigarettes per day, practically identical to the 15.4 percent reported when contacted by mail 12 months earlier.

Among the mail responders, men who had quit or reduced their smoking were older than those who continued smoking, and had smoked somewhat fewer cigarettes but had smoked longer than those who continued smoking. Initial CO_a levels were significantly lower in quitters than in those whose smoking was decreased or unchanged. Prevalence of asbestosis was over twice as high in those who quit or cut down as compared to those who continued. The lowest mean pulmonary function values and the most pack years of smoking were in men who decreased their smoking but had not quit; they also had more airways obstruction, more air trapping, and lower vital capacities.

The telephone interviews of 101 non-responders showed that 17 percent had quit and remained non-smokers, another 2 percent were recidivists, 53 percent had reduced cigarettes smoked per day, and 28 percent continued smoking as before (Table 3). In the telephone sample, fewer had quit but more had reduced their smoking when compared to the mail sample; of the 34 men with asbestosis, 12 (35 percent) had quit and 17 (50 percent) had reduced their smoking.

Discussion

Quit rates of 17 percent by telephone interviews of non-responders and 30 percent by mail questionnaire were 3.6 to 6.4 times the quit rate of 4.7 percent in the previous year and much above the 2.5 percent to 5.5 percent in the preceding 20 years. Thus, for this blue collar population, the demonstration of their examination, lung function, and x-ray findings and personal advice about present or future health problems from smoking appear to have motivated many of them to quit or reduce their smoking.

The difference in quit rates between the men who responded by mail (30 percent) and non-responders contacted by telephone (17 percent) was not surprising. A 19 percent response is not unreasonable considering the interval of 6–25 months after previous contact. Those with a more socially acceptable outcome, i.e. quitting, would be expected to reply more frequently. Nevertheless, if one applies the 17 percent quit rates of those reached by telephone to the entire

TABLE 3—Categories of Smoking in 101 Telephone Interview Responders by Diagnosis of Asbestosis and Initial Avelolar Carbon Monoxide Level

	Smoking Status				
	Quit	Reduced	Continued	Recidivist	Total
Age years	53.7	51.6	51.6	53.6	
No asbestosis	5	36	24	1	66
Initial CO_a ppm	17.1 ± 8.9	38.5 ± 14.9	34.3 ± 14.1		
Asbestosis	12	17	4	1	34
Initial CO_a ppm	36.1 ± 13.1	33.6 ± 16.9	28.7 ± 14.4		
Total	17	53	28	2	100

mail non-responder group and adds the 171 who quit in the mail responder groups, 531 men are estimated to have quit. This is 20 percent of the 2,627 men screened, at least twice the quit rate of the previous year even if the annual quit rate of 4.7 percent is doubled to make the time intervals comparable.

If decreasing smoking indicates progress toward quitting, then a major proportion (36 percent to 53 percent) of the men were influenced by the intervention. This is supported by the finding in the telephone follow-up that 23 percent of those who had earlier decreased their smoking, quit 12 months later.

Reliability of self reporting has been a controversial subject. Some investigators,[12] have found 95 percent accuracy for telephone interviews compared to personal interviews except for short-term quitters. This reservation would not apply to the long-term quitter. Furthermore, it should be realized that these men did not receive strong or continuous external pressure to quit. They were not even aware, until follow-up, that the intervention was being evaluated. The high correlation between smoking histories and CO_a in the initial study suggested that misleading self-reports about smoking were infrequent.

It is unlikely that the clear differences between quit rates of less than 5.5 percent per year prior to intervention and the 17 percent to 30 percent of new quitters could be due to bias introduced by either the mailed questionnaire or the telephone inquiry. Follow-ups by telephone to those who had responded by mail confirmed their quitting. Veracity was further strengthened by a reported 3.6 percent recidivist rate.

Our intervention was less intense than that in a group of British civil servants who, in hospital because of symptoms or signs of coronary disease or chronic bronchitis or both and received individual advice, were challenged to "consider their situation" and given support and encouragement during four further visits over a period of 12 months. That study had 63 percent of responders (84 percent of the group) who reported being off cigarettes after one year. The present project was also less intensive than a Mayo clinic study of motivated volunteers, mostly referred by physicians in 1975 and seen annually through 1984. They received eight weekly one-hour sessions including presentations from an ear, nose and throat specialist and from a pulmonary physician and achieved a 22.3 percent quit rate at one year,[14] results similar to ours. Nevertheless, our quit rate of 17 to 30 percent of the current smokers plus 36 percent who decreased by 15 cigarettes per day was a substantial beginning.

The men we studied had not volunteered for smoking cessation but were recruited through their unions because of asbestos exposure. Thus, their concerns were whether they had asbestosis, pleural changes, or pulmonary disease. The objective of our intervention was to motivate them to quit smoking, not to help self-motivated volunteers to quit. It appeared to us that the most important motivating factor was measurement of CO_a. Although CO_a had been used previously as a reliability check on smoking histories,[12] in our study it quickly showed these workers a smoking "effect." The smokers' carbon monoxide level was a "big number," in parts per million, compared to that of a nonsmoker or ex-smoker. After CO_a, the two next most powerful motivators seemed to be the x-ray appearance of asbestosis and airways obstruction on the spirogram. Because each individual was "targeted" with personal information, it was a novel experience with strong impact.

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Epidemiologic Differences between Chlamydia and Gonorrhea

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Abstract: To assess the prevalence, demographics, and transmission patterns of genital chlamydia infection, we screened 3,078 patients, and compared identified cases (N = 511) to gonorrhea cases (N = 291) diagnosed in the same setting. Chlamydia cases were younger and more likely to be White than their gonorrhea counterparts. Chlamydia cases were distributed diffusely; geographic overlap between the two diseases was only about 40 percent. Gonoccocal coinfection was noted in <10 percent of patients with chlamydia. Nearly half of men with chlamydia and four-fifths of women were asymptomatic and most cases were identified through screening or contact tracing. Populations at high risk for chlamydia are seemingly different from those for gonorrhea. Differences may be due to control interventions (active for gonorrhea, passive for chlamydia). Chlamydia case reporting and control initiatives are recommended. (*Am J Public Health* 1990; 80:1338–1342.)

Introduction

Chlamydia trachomatis causes an estimated three to four million infections annually in the United States; its morbidity, particularly salpingitis, is substantial.[1,2] Nevertheless, only about half of the states require its notification to public health officers.[3] Although much has been learned about its etiology and clinical aspects in the last two decades, little has been published about its epidemiology. Until recently, reliable and inexpensive screening tests have not been available.

To develop a sense of chlamydia prevalence, patient characteristics, and transmission patterns, we implemented a year-long investigation during 1988. Unlike gonorrhea, chlamydia is not a reportable disease in Colorado. Thus we present epidemiologic and case-finding observations on cases of genital chlamydia infection that were detected in the public medical sector, and we contrast some of these findings with gonorrhea cases ascertained in the same setting. In addition, we were able to obtain comparative data from a local military post, where chlamydia and gonorrhea surveillance information is routinely collected.

Methods

Demographics

The study site is Colorado Springs, a standard metropolitan area (SMA) with a population of about 400,000 (1988 estimate), 65 miles south of Denver. Ethnically, the population is 83.5 percent White, 6 percent Black, 8.2 percent Hispanic, and 2.3 percent other races; it is about two years younger than the national median of 31.8 years (1986 estimate), principally because of the large military population. The military post hosts 60 percent of the 35,000 active duty military personnel in the SMA, 92 percent of whom are men. The post population is 65 percent White, 27 percent Black, 4 percent Hispanic, and 4 percent other races. The public clinics are in the SMA's only health department.

Case Definition

The definitive diagnosis of genital chlamydia infection is based on performance of a moderately difficult and expensive cell culture isolation technique. Because this technique was beyond our means, identification of cases and testing of their sexual partners in both the public and military sectors was done with a less reliable but affordable test (MicroTrak, Syva Laboratories, Palo Alto). Hence all cases in our study are presumptively diagnosed.

MicroTrak is a direct immunofluorescence assay using monoclonal antibodies that react with all 15 known human chlamydia serovars; it was performed according to the manufacturer's instructions. Three test outcomes are possible: positive, suspicious (the presence of fewer than 10 chlamydial elementary bodies), and negative.

Test Sensitivity

Because the performance characteristics of MicroTrak were not evaluated by the "gold standard" (McCoy cell culture) in our clinical settings, test outcomes may differ from published reports; these usually claim that the sensitivity of a single genital attempt to isolate *Chlamydia trachomatis* by the McCoy cell culture is about 90 percent for men and between 70 percent and 80 percent for women,[4] and MicroTrak's sensitivity compared to cell culture is about 70 percent for men and 90 percent for women, with a specificity of about 98 percent.[5] Therefore, MicroTrak's ability to detect genital chlamydia in men approaches two-thirds, a value almost identical to the one for women. We use two-thirds as a proportion to estimate the shortfall in our case-finding outcomes.

Patient Selection

Clients presenting for genital examination at our health department STD, prenatal, or family planning clinics were screened for chlamydia during calendar year 1988. Gonorrhea screening in these clinics is universal, except in the family planning clinic, where the gonorrhea culture is used for diagnosis or clinical suspicion and results in about 17 percent of clients being tested. Budget constraints permitted chlamydia screening of only half of the clients in the STD clinic: 52.5 percent of 1,546 women and 55.6 percent of 1,657 men. Although STD clinicians were encouraged to screen younger women (under age 25), all sexual partners of patients with genitourinary conditions and all patients with recurring genitourinary symptoms, the clinicians were not bound by

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these recommendations. All clients in prenatal (573) and all initial visitors to family planning clinic (772) were screened.

Case-finding

Symptomatic men were asked to identify all sexual partners in the interval from one month before the appearance of their urethral symptoms to the date of their treatment; asymptomatic men and all women were queried about their partners in the 120 days prior to the date of their positive test. In the event that there was only one sexual partner in that interval, these latter patients were asked to identify at least one more sexual partner during the year before their diagnosis. The sexual partners (contacts) were actively sought by encouraging self-referral and through field follow-up. All traced contacts were offered epidemiologic treatment as recommended by the Centers for Disease Control (CDC).[6]

Geographic Distribution

The concentration of cases was mapped; census tracts containing approximately 50 percent of cases were considered Core, those containing the next 30 percent, Adjacent, and the remainder, Peripheral, according to Rothenberg's method.[7]

Data Management

Information was recorded on medical charts, case interview records (CDC form 9.97), and contact investigation reports (CDC form 9.2936). The data were coded and entered, without personal identifiers, onto a VAX 8600 computer (Digital Equipment Co.), and they were analyzed using a standard statistical package (SPSS-X, version 3.1).

Results

Population Selection

Younger clients in STD clinic were likely to be tested for chlamydia; the mean age of those tested was 25.3 years (95% CI = 25.0, 25.6) compared to 26.7 years (95% CI = 26.3, 27.1) for untested clients. No tendency to preferentially test by gender or ethnicity was noted.

There were no demographic or case-finding differences between positive and suspicious cases of chlamydia in men; all are included in this report as probable cases. Clinically, 62 percent of the 45 suspicious cases in men were asymptomatic or minimally symptomatic; 19 presented as sexual partners of known cases and 11 had their infection detected as a consequence of screening. The corresponding percentages in the 198 positive cases were: 41.9 (N = 83), 31.3 (N = 62), and 6.6 (N = 13). Concomitant gonococcal infection was observed in 8.6 percent of the positive cases, but in none of the suspicious cases.

Positive and suspicious chlamydia cases in women differed with respect to age, reason for presentation, and case-finding outcomes. Suspicious cases (N = 38) were more likely to be older, with a mean of 25.5 years (95% CI = 23.5, 27.5) versus a mean of 20.7 for positive cases (95% CI = 20.2, 21.2); to present as a result of perceived genitourinary symptoms (39.5 vs 19.8 percent) rather than as contacts or as screenees, and to have fewer chlamydia infected partners in their sexual environments (19.2 percent vs 35.5); 19.2 percent is close to the background level of chlamydia infection in the sexual partners of women who present in our public clinics. Therefore, suspicious cases in women were deemed possible, rather than probable, cases and excluded from the analysis.

Our final study population consists of the 511 cases of chlamydia classified as probable cases in 1988. There were 243 cases in men (198 positive and 45 suspicious cases), all from the STD clinic, and 268 cases in women: 132 from the STD, 75 from prenatal, and 61 from family planning, clinics.

Age Distribution

Sixty percent of the 243 chlamydia cases in men (mean age = 24.2; 95% CI = 23.5, 25.0) and 84 percent of the 268 cases in women (mean age = 20.7; 95% CI = 20.2, 21.2) are under age 25. Fully half of the women are teenagers, as are 20.7 percent of the men. In contrast, 47.5 percent of the 139 gonorrhea cases in men (mean age = 26.1; 95% CI = 24.8, 27.4) and 70.4 percent of the 152 gonorrhea cases in women are under age 25 (mean = 22.9; 95% CI = 21.9, 23.9). Fifty-six percent of men with gonorrhea are in their twenties, whereas 53.2 percent of women are between ages 17 and 22.

Similar differences are noted for the military cases: 80 percent of the 238 chlamydia cases in men whose age was available (vs 72.4 percent of the 337 gonorrhea cases) and 81.7 percent of the 181 age-available cases in women (vs 68.1 percent of the 91 gonorrhea cases) are under 25 vs over 25 years old. The younger distribution in military cases probably reflects the younger age of such populations. The age distribution, by gender, of the sexual partners of our chlamydia patients was similar to that in cases.

Ethnic Distribution

Representation by Whites is higher in chlamydia than in gonorrhea cases for both men and women (Table 1). A similar pattern is noted for military cases, even though the aggregate venereal burden is overwhelmingly Black (Table 2). The race

TABLE 1—Comparison of Ethnic Distribution of Public Clinic Chlamydia and Gonorrhea Cases, by Gender, Colorado Springs, 1988

	Ethnicity			
Cases	White	Black	Hispanic/Other	Total
Men	N (%)	N (%)	N (%)	N (%)
Chlamydia†	150 (61.7)	60 (24.7)	33 (13.6)	243 (100)
Gonorrhea	56 (40.3)	73 (52.5)	10 (7.2)	139 (100)
Women				
Chlamydia‡	161 (60.1)	44 (16.4)	63 (23.5)	268 (100)
Gonorrhea	71 (46.7)	67 (44.1)	14 (9.2)	152 (100)
Total	438	244	120	802

†$\chi^2 = 30.41$; DF = 2; $p < 0.00001$ for the ethnic difference between chlamydia and gonorrhea in men.
‡$\chi^2 = 42.03$; DF = 2; $p < 0.00001$ for the ethnic difference in women.

TABLE 2—Comparison of Ethnic Distribution of Military Chlamydia and Gonorrhea Cases, by Gender, Colorado Spring, 1988

	Ethnicity			
Cases	White	Black	Hispanic/Other	Total
Men	N (%)	N (%)	N (%)	N (%)
Chlamydia†	92 (40)	120 (52.2)	18 (7.8)	230 (100)*
Gonorrhea	52 (15.4)	270 (80.1)	15 (4.5)	337 (100)
Women				
Chlamydia‡	85 (46.7)	82 (45.1)	15 (8.2)	182 (100)**
Gonorrhea	29 (31.9)	51 (56)	11 (12.1)	91 (100)
Total	258	523	59	840

†$\chi^2 = 50.71$; DF = 2; $p < 0.0001$ for the ethnic difference between chlamydia and gonorrhea in men.

‡$\chi^2 = 5.14$; DF = 2; $p < 0.1$.

*Data not available for 20 cases.

**Data not available for 15 cases.

distribution, by gender, of the sexual partners to our chlamydia patients was similar to that of cases.

Geographic Concentration

In the Colorado Springs SMA, the observed chlamydia cases are more diffusely distributed than gonorrhea cases, and the overall extent of geographic overlap between the two diseases is approximately 40 percent. These differences are mapped in Figures 1 and 2. Whereas 50.5 percent of our overall chlamydia cases are observed in 11 Core census tracts (16.2 percent of the SMA's 68 census tracts), nine (11.8 percent) census tracts comprise the gonorrhea Core (49 percent of cases). Only six of the 14 combined chlamydia and gonorrhea Core census tracts (42.9 percent) overlapped. The Adjacent distribution of chlamydia was more diffuse: 29 percent of chlamydia cases occupied 20 census tracts, versus 29.3 percent of gonorrhea cases which occupied 14 census tracts. Overlap occurred in only nine (36 percent) of the 25 combined chlamydia and gonorrhea Adjacent tracts.

The geographic distribution of the sexual partners of chlamydia patients was similar, except that fully 10 percent of contacts emanated from a single military census tract (where *cases* were not enrolled for this study). Exactly two-thirds of all sexual partners resided in Core/Adjacent census tracts, as opposed to about 80 percent of cases. However, the geographic distribution of infected contacts mirrored that of cases: 47.6 percent resided in the Core, and 37.8 percent in the Adjacent, census tracts.

Clinical Findings

One-half (57 percent) of the chlamydia cases in men presented because of symptoms (54 percent had frank urethritis). The remainder were identified through contact tracing (33 percent) or screening (10 percent); these men were essentially asymptomatic.

Chlamydia cases in women were identified through contact-tracing (20.5 percent) or through screening (59.7 percent); only about one-fifth presented spontaneously for examination.

Of the 2,437 public clinic clients tested simultaneously for both infections, 419 had chlamydia, 93 gonococci, and 41 both organisms. Thus 9 percent of chlamydia patients, and 31 percent of gonorrhea patients, were infected with both organisms.

Case-Interviewing

About four-fifths of chlamydia cases were interviewed for sexual partner information (Table 3). No case named a sexual partner of the same gender. The case-interview yield was similar to that obtained in gonorrhea cases.

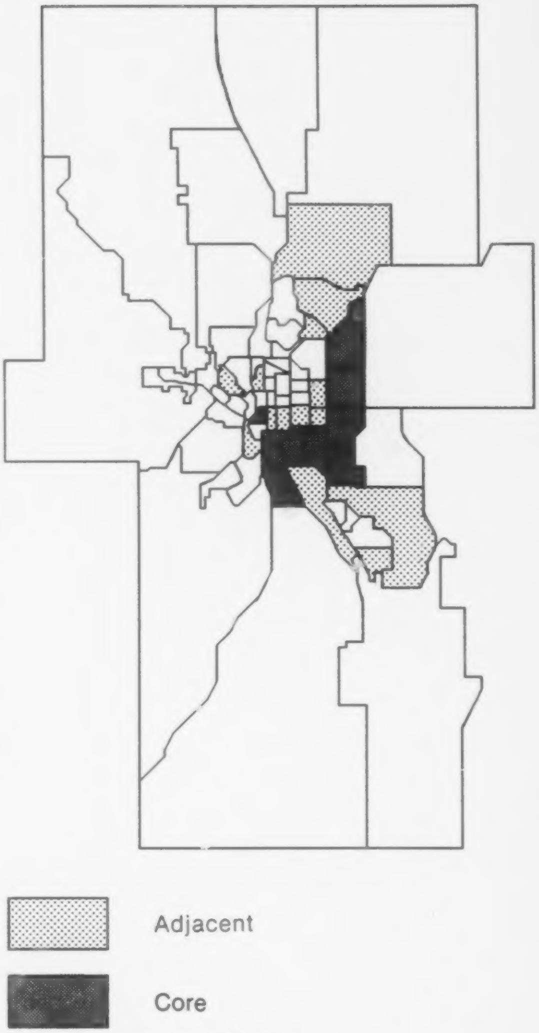

FIGURE 1—Gonorrhea Case Distribution
Public clinic cases (N = 291) in Colorado Springs in 1988: Census tract map showing core and adjacent areas (See Methods)

Had perfect tests been available to use on all examined contacts, the proportion infected would have been roughly the same for contacts to each disease, with chlamydia contacts exhibiting a slightly higher rate of infection in both men and women.

No ethnic differences were noted between chlamydia cases and their contacts.

Case-finding Efficacy

A total of 94 sexual partners had their infection newly identified as a result of case-interviewing and contact tracing (40 female and 54 male contacts). This result produces a 0.22 new case yield (94/419 interviews). Had perfect tests been available, an estimated 141 new cases would have been

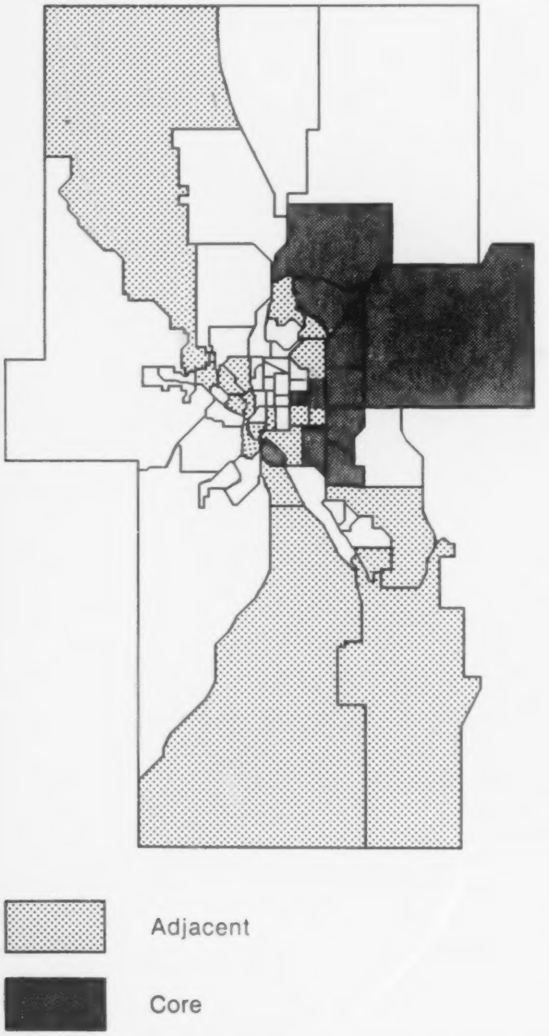

FIGURE 2—Chlamydia Case Distribution
Public clinic cases (N = 511) in Colorado Springs in 1988: Census tract map showing core and adjacent areas (See Methods)

identified, for a yield of 0.34. Thus, we estimate that public health workers need to interview three chlamydia cases and seek their sexual partners (about five), to remove one previously untreated, infected person from the disease pool.

Self-Referral Behavior

Of the 326 named sexual partners who newly were eligible for examination and were found, only 102 (31 percent) were successfully referred by the original chlamydia patient; the other 224 sexual partners required active intervention on our part to assure presentation. Of the 94 newly identified infected contacts, 34 (36 percent) were partner-referred. Black men were least likely to be partner-referred (17 percent), White men most likely (40 percent); Hispanic/Other men, 24 percent. Women were equally likely to be partner-referred (about 22 percent), irrespective of ethnicity.

Discussion

Although ours is not a population-based study of chlamydia prevalence, we found demographic differences between public clinic patients with chlamydia and those with gonorrhea, yet these two diseases infect columnar epithelial tissue and share the same mode of transmission. In Colorado Springs during 1988, public clinic cases of probable genital chlamydia and gonorrhea infection differed with respect to age, ethnicity, and domicile; clinically, little overlap between the two diseases was noted. The same is true for their sexual partners.

Sources of bias in the present study include testing and patient selection. Although neither the sensitivity nor specificity of the MicroTrak test was established locally, it is noteworthy that the estimated proportions of infected persons per sexual partner examined are nearly identical to those obtained in Sweden.[10] If the Swedish data are adjusted for the relative insensitivity of the McCoy cell culture test used in that study, we arrive at 0.84 (estimated) infections identified per Swedish woman contact examined and 0.61 per Swedish man contact, compared to 0.87 and 0.56 (estimated) respectively in our study (Table 3).

The tendency to test younger clients in STD clinic probably affected the age distribution of chlamydia cases. This bias is not likely to be substantial: age differences were also noted in the military cases we reviewed and these were not differentially selected. Bias stemming from incomplete testing for gonorrhea in the family planning clinic probably is minimal: characteristics of gonorrhea cases in this study closely match those from a previous report.[11] The insensitivity of chlamydia testing in asymptomatic men would create bias only in the case of differential insensitivity, which is not likely.

The typical chlamydia case tended to have infection detected as a consequence of screening or contact tracing, rather than via spontaneous presentation with symptoms. It is possible that asymptomaticity is one clue to observed differences in the community expression of these two diseases. In our experience men were at least twice as likely to be asymptomatic with genital chlamydia than with gonorrhea,[8] a ratio similar to that reported in Sweden.[10]

It is possible that sustained, vigorous gonorrhea control efforts on our part since the mid-1970s[12,13] influenced the observed distribution of chlamydia cases in our community. In Colorado Springs this population is principally White. As our interventions helped to decrease the gonorrhea burden from a high of about 2,000 cases in 1977 to about 1,000 cases in 1988, the proportion of cases accounted for by non-Whites increased from 39 percent in 1977 to 67 percent in 1988. This phenomenon may also explain the modest proportion of clinical overlap between the two diseases since gonorrhea is relatively well controlled in Whites locally.

We have demonstrated previously that gonorrhea is neither randomly nor evenly distributed in our sexually active populations[11] and that the basic setting for sexual aggregation and for sexual choices is the neighborhood. It is possible that in neighborhoods where the gonorrhea burden is high, the chlamydia burden may be lower than expected because of frequent exposure to antibiotic treatment on the part of gonorrhea cases and their sexual partners, especially since the introduction of dual treatment for gonorrhea and chla-

TABLE 3—Chlamydia Case—Interview Outcomes in 1988, Compared with Previously Reported Gonorrhea Case-Interviews in Colorado Springs, by Gender

Cases						
Disease	Interviewed	Contacts Elicited	Contacts/ Case	Examined (%)	Infected‡ (Observed)	Infected** (Expected)
Men				Contacts (Women)		
Chlamydia	190	321	1.7	176 (55)	98 (.58)	147 (.87)
Gonorrhea*	394	827	2.1	533 (64)	381 (.71)	423 (.79)
Women				Contacts (Men)		
Chlamydia	229	379	1.7	254 (67)	94 (.37)	141 (.56)
Gonorrhea†	578	1492	2.6	1042 (70)	393 (.38)	491 (.47)

*Data from reference 8
†Data from reference 9
‡Per contact examined
**Expected if laboratory tests were perfect. For chlamydia we use two-thirds as the observed sensitivity for men and women (see Methods) and for gonorrhea, 90% sensitivity for men and 80 percent for women.

The infection rate for women contacts to men with chlamydia may be underestimated because 21 of the 176 "examined" women were treated by their private physician without being tested.

mydia in the early 1980s.[6] Conversely, in neighborhoods where gonorrhea is an infrequent event, exposure to chlamydia may result in successful infection more often. In addition, entrenchment and perpetuation of chlamydia may be facilitated by its high rate of asymptomaticity. Age differences between chlamydia and gonorrhea patients may be accounted for by a tendency on the part of teenage women with chlamydia to select sexual partners close to their own age.

More research is needed to elucidate the factors that contribute to differences in the community distribution of these two closely related diseases. Meanwhile it is clear that public health intervention is crucial to the control of chlamydia infection in Colorado Springs. The disease is frequently asymptomatic, occurs often in populations that we are not encountering routinely in our standard STD outreach efforts (syphilis, gonorrhea, and human immunodeficiency virus infections), and the chlamydia-prone populations are only moderately successful at referring their sexual partners. Our data support a recent appeal to implement a "program of chlamydia control that is based on accurate diagnosis, contact tracing, appropriate therapy, and educational efforts to reduce risky behavior . . ."[14] Our first step is to encourage the reporting of chlamydia cases to public health officers.

ACKNOWLEDGMENT

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A Small Area Simulation Approach to Determining Excess Variation in Dental Procedure Rates

PAULA DIEHR, PHD, AND DAVID GREMBOWSKI, PHD

Abstract: All small area analyses need to compare the observed variability in rates to that expected by chance alone, but the expected variability is usually not known. This paper uses patient-level data for five dental procedures to simulate the distributions of the summary statistics that are usually generated in such studies. These statistics are found to vary greatly even under the "null hypothesis" that all dentists are using procedures at the same rates. The simulated dentist rates are compared to observed rates obtained in a different study. These findings illustrate problems that can occur in small area analysis studies, and emphasize the importance of using statistical techniques that are appropriate for the data that are to be analyzed. Investigators should make every effort to obtain patient-level data, or at least to understand the underlying distribution of the number of procedures per patient, to avoid mistaking significant deviations from an incorrect model as evidence for significant variation among small areas. (*Am J Public Health* 1990; 80:1343–1348.)

Introduction

Small area analysis is a popular methodology in health services research. A typical study might calculate the utilization rate for a service in several small areas, compare the largest rate to the smallest, note that the difference is large, and attempt to explain the high variability as a function of service availability, physician uncertainty, and other variables of interest. The statistical methods used in such studies have been questioned by an author of this paper.[1]

A recent paper using computer simulation to examine the statistical properties of the most frequently used descriptive statistics[2] demonstrated that all of the usual descriptive statistics could be deceptive, showing large apparent variation when there was no more variation than would be expected by chance alone. A simple 2 × k chi-square test (classifying people in each of k communities into two cells, by whether they had or did not have the procedure) was an appropriate test for excess variability as long as each person could have the procedure at most once. However, the chi-square test could be deceptive; results would be "statistically significant" too often if an individual could have the procedure more than once. Simulation studies were recommended for these situations; this requires that the distribution of the number of procedures per person be known. Finally, it was pointed out that studies very similar to small area analyses were being conducted in other situations: e.g., a "small area" might be a hospital or a dental practice. This paper applies the small area simulation approach to a set of dental data, to determine if there is more variation in procedure rates among dentists than would be expected by chance alone.

Grembowski, *et al*,[3] calculated procedure rates for 200 dentists in general practice in four urban counties of Washington State from 1984 and 1985 dental claims of members of the Washington Education Association (WEA) and their dependents. The number of patients seen at least once in the two-year period ranged from 75 to 300 for the 200 dentists, as shown in Table 1.

Procedure rates for each dentist were calculated by dividing the total number of procedures performed in a two-year period by the number of people seen at least once by the dentist during that period. Results were age-adjusted. Table 2 shows the substantial variability that was found among dentists: rates of oral examinations ranged from 10 per 1,000 patients to 3,543 per 1,000 patients. The coefficient of variation (the standard deviation divided by the mean) was 0.289. This seems like substantial variation. However, a good deal of variation can be expected by chance alone, even if the underlying rate of examinations per person is the same in each practice.[2] If "exams" were a binary variable, which a person could have once or never, it would be appropriate to use a 2 × 200 chi-square test to examine the null hypothesis that the probability of having an oral exam was the same in all practices.[2] However, it is obvious that many people have more than one examination in two years, which means that the chi-square test and the usual small area statistics are either inappropriate or have unknown distributions under the null hypothesis.

TABLE 1—Number of Washington Education Association Patients Seen by each Dentist in Two Years

N of Patients Seen	N of Dentists
75–99	94
100–124	54
125–149	19
150–199	24
200–300	9

If the number of oral exams for each person were known, techniques could be developed to study whether there was excess variation. The data could be recoded so that each

TABLE 2—Observed Procedure Rates in 200 Dental Practices (number of procedures per 1,000 patients)

Category	Mean	SD	Min	Max	EQ	CV
Oral Exams	1617	467	10*	3543*	354.3*	.289*
Fillings	889	373	188*	2379	12.65*	.420*
Extractions	119	83	0	523	—	.698*
Root Canals	44	40	0	228	—	.909*
Scalings	23	47	0	345*	—	2.044*

EQ = Maximum/Minimum.
CV = coefficient of variation.
*Minimum lower than 5th percentile of null distribution, or maximum higher than 95th percentile (see Tables 3 and 4).

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person was coded 1 if he had one or more exams, and 0 otherwise, and the chi-square statistic could be used; or, one could assume that the number of exams per person was approximately normally distributed, and perform an analysis of variance to study whether the average number of exams per person were different among the dentists. Unfortunately, the only data available for the Washington study were the rates per dentist of the various procedures. No patient level data were available. For these reasons, we attempted a simulation approach, discussed elsewhere in detail.[2] The goal is to determine what the distribution of procedures among the 200 practices would have been under the null hypothesis that no dentists were "high users" but, on the contrary, that all dentists performed the same number of procedures per person, on average. The simulation method attempts to determine how much variation among practices would be expected by chance alone. This is then used as a benchmark to determine whether the observed variation in Table 2 is important.

Methods

Data

A different data source was used to obtain information about the distribution of procedures at the patient level. Annual dental utilization data were obtained for 12,897 insureds from a random sample of adults and children covered by Pennsylvania Blue Shield dental insurance in 1980. These data have been analyzed elsewhere.[4,5] The 5,912 people with no dental visits in 1980 were eliminated from the data set. Appendix 1 contains frequency distributions for five dental procedures—oral exams, fillings, simple extractions, root canals, and full-mouth periodontal scalings. (For example, 2,764 patients had no oral exams, 2,717 had one exam, and six people had 12 oral exams in one year.)

Fitting the Pennsylvania Data

We first attempted to fit the Pennsylvania data to the Poisson and negative binomial distributions.[6] If a known distribution was appropriate, we could use it to generate random data for the simulation. Clearly, none of the data are normally distributed because of the long tails and the high number of observations clustered at zero. The Poisson distribution is not likely to fit the data because a Poisson process assumes that procedures occur independently, which is probably not the case for dental procedures. The negative binomial distribution assumes that each person uses procedures with a Poisson distribution, but that each person has a different Poisson parameter, distributed according to a gamma distribution.[6] This distribution was considered because it has longer tails than the Poisson distribution.

We estimated the parameters of the Poisson and negative binomial distributions using the maximum likelihood procedure.[6] This yielded expected numbers which were compared to the observed numbers with the chi-square goodness of fit test.[7] Neither distribution provided an acceptable fit for the number of oral exams or for fillings. The negative binomial distribution did provide an adequate model for the number of extractions, root canals, and scalings.

The following simulation work did not assume any mathematical distribution, because there was none that worked for all the data. Rather, data were generated directly from the empirical distributions, as described below. Future work in simulating procedure rates for extractions, root canals, and scalings might use the negative binomial distribution to generate random data, however.

Simulation of Utilization Rates

Our goal was to develop a simulation model to assess the amount of variability that would have occurred among the dental practices by chance alone if the null hypothesis (that all dentists use procedures at the same underlying rate) is true. We generated 100 different simulated sets of procedure rates, in which the null hypothesis was true.

For each of the 200 dentists, a uniform random number between 0 and 1 was generated for each patient in that practice (e.g., for a dentist with 75 patients, 75 random numbers were generated). Each of these numbers was compared to the cumulative distribution in Appendix 1. For example, 2764/6985 or 39.5 percent of the people had no oral exam; 78.46 percent had 0 or 1 exam; 97.7 percent had 0, 1, or 2 exams, etc. For each random number, if it was less than .395, the patient was assigned 0 exams; if between .395 and .7846, the patient was assigned one exam, etc. Thus, on average, the number of exams per patient match the distribution of Appendix 1, but the distributions were somewhat different for each dentist.

After the number of exams were generated for all 75 patients in the first practice, the exams were summed, multiplied by 2 (see discussion section) and divided by 75 to provide a simulated two-year rate of oral exams for that dentist. This was then continued for all 200 dentists, which generated 200 different simulated examination rates. Next, the minimum rate, the maximum rate, the extremal quotient (EQ, ratio of the maximum rate to the minimum), the coefficient of variation, and other statistics explained in the results section were calculated. This process was repeated 100 times to give, for example, the "average" maximum rate, or the 95th percentile of the maximum rate, under the null hypothesis that each practice has the underlying distribution of examinations shown in Appendix 1.

Results

Mean Procedure Rates

Table 2 shows the observed results, and Table 3 shows the simulated results. The first column in Tables 2 and 3 is the estimated mean number of procedures per 1,000 patients. Recalling that the two sets of numbers arise from different data sets, there is fairly good correspondence on the rate of oral exams (1,725 versus 1,617), root canals (55 versus 44), and full mouth scaling (34 versus 23). There is very poor agreement on fillings (2,123 versus 889) and for simple extractions (211 versus 119). This may be due to differences in billing practices between Pennsylvania and Washington Dental Service—the carrier for Washington Educational Association. There are probably also socioeconomic differences between the Washington and the Pennsylvania patients. These differences are considered further below.

TABLE 3—Simulated Procedure Rates (number of procedures per 1,000 patients)

	Rate Mean	Minimum		Maximum		EQ	
		Mean	5%	Mean	95%	Mean	95%
Oral Exams	1725	1260	1136	2304	2537	1.8	2.1
Fillings	2123	1195	974	3422	3897	2.9	3.4
Extractions	211	30	0	529	667	17.5	30.1
Root Canals	55	0	0	259	342	—	—
Scalings	34	0	0	227	316	—	—

EQ = Maximum/Minimum
Based on 100 iterations per procedure.

Maximum and Minimum

The simulated minimum and maximum are shown in Table 3. For example, on average, the lowest rate of oral exams was 1,260 per 1,000 patients, and the highest rate was 2,304 per 1,000, even though the underlying rate for all dentists was 1,725. Thus, an observed minimum rate near 1,260 or a maximum rate near 2,300 would not be an indication of excess variability among practices. Table 3 also shows the simulated 5th percentile of the minimum and 95th percentile of the maximum. That is, 95 percent of all minimum values are above 1,136, and 95 percent of all maximum values are below 2,537.

The observed minimum rates for oral exams, shown in Table 2, can be thought of as extremely low, since 10 is substantially below 1,136. The same is true for fillings. The minimum for the other procedures is 0 for both the observed and the simulated data. Comparing the maximum values of Table 2 to the 95th percentiles in Table 3, the maximum rates for oral exams and for full mouth scaling are higher than would be expected by chance alone, but the other maxima are not particularly high.

Extremal Quotient (EQ)

Table 3 shows the simulated average value and the 95th percentile of the EQ (maximum divided by minimum). For oral exams, one would expect an EQ of about 1.8, and values above 2.1 (the 95th percentile) would be considered particularly high. For fillings, a ratio of 2.9 is expected, and 3.4 would be considered high For extractions the minimum value was 0 in 10 percent of the simulations, and the EQ could not be computed. For the remaining 90 percent, the expected EQ was 17.5 and the 95th percentile of the EQ was 30.7. The EQ is infinite for root canals and scalings, as the minimum is always zero. In Table 2, the observed EQ could be computed only for oral examinations and fillings. Both of the observed EQs are much larger than the 95th percentile of the EQ distribution, indicating that the null hypothesis that all dentists had the same rates is probably not true. The variability in the rates is due in part to some dentists having much *lower* use of procedures than would be expected.

Coefficient of Variation (CV)

Table 4 shows that the 95th percentile of the simulated CV (standard deviation over mean) for oral exams is 0.115, as compared to an observed value of 0.289 shown in Table 2. The observed variation is thus higher than would have been expected by chance alone. All of the observed CVs are considerably above what would have been expected. Therefore, the null hypothesis probably is not true, and there are some dentists who use procedures at different rates from the others.

TABLE 4—Simulation Results—Other Summary Statistics*

	95th percentiles (per 1,000 patients)				
	CV	SCV	199 df Chi sq	1 df Chi sq	F
Oral Exams	.115	8	−441	7.28	1.22
Fillings	.194	35	−1116	27.31	1.13
Extractions	.442	148	1070	13.15	1.17
Root Canals	.914	674	1035	14.35	1.19
Scalings	1.188	1468	1208	18.97	1.20
Degrees of Freedom			199	1	199, inf
Tabled 95th Percentile			233	3.84	1.17

*CV is the coefficient of variation and SCV is an estimate of the systematic component of the variation, defined in the text. Chi-square with 199 degrees of freedom is 2 × k chi-square assuming that each procedure is a person. Chi-square with 1 df is the test for a particular practice (see text). F is the F statistic that arises from a 1-way analysis of variance. Tabled 95th percentiles are taken from tables of the chi-square and F distributions.

Based on 100 iterations per procedure.

Systematic Component of Variation (SCV)

Table 4 shows some other statistics commonly used in small area analysis, which were not calculated for the Washington data. They are included for completeness. The SCV has been proposed by McPherson and Wennberg as a test for excess variability among small areas.[8] The formula is:

$$SCV = (1/k)\ [\Sigma((O_i - E_i)^2)/E_i^2 - \ \Sigma(1/E_i)]^*\ 1000.$$

There is an F-test associated with this statistic, but it is not often used. The SCV was derived under the assumption of Poisson rates, which is not appropriate here. The data in Table 4 illustrate further what has been shown elsewhere[2]; that is, that the SCV can take on very large values, even when there is no underlying variation among the small areas. In addition, it becomes larger as the mean rate becomes smaller. Thus, it would be unwise to use the SCV alone as a descriptive statistic in this situation.

Chi-Square

Two chi-square tests have been used in small area analyses. One test is a 2 × 200 chi-square test, which assumes that each person received a procedure at most once, calculates the number not receiving the procedure as "population size minus the number of procedures," and creates a 2 × 200 table. This is clearly inappropriate in our situation, since many people had more than one procedure. In fact, the 95th percentile of this chi-square statistic is negative for oral exams and fillings, because the expected numbers of people who did not have the procedure are negative for some dentists (for example, there are more fillings than patients). A chi-square value greater than 1,000 would be needed to demonstrate "significance" for the last three procedures, as compared to the tabled 95th percentile of the chi-square with 199 degrees of freedom, which is 233.

Another test which is proposed in these situations is a 1 degree of freedom chi-square test of the hypothesis that a particular dentist has a rate significantly different from what would have been expected. The expected number of procedures would be the grand rate (e.g., 1,725 oral exams per 1,000) times the number of patients (e.g., 75, for an expected rate of 129 exams). If the observed rate were, e.g., 155, the chi-square statistic would be $X = (O - E)^2/E = (155 - 129)^2/129 = 5.2$. This is much higher than the usual critical value of 3.84 for the chi-square distribution with 1 degree of freedom. However, Table 4 shows that the 95th percentile of chi-square statistics in this situation is 7.28, meaning that 5.2 is not a particularly large value. The 95th percentiles for the other dental procedures are even larger, ranging from 13 to 27. Thus, the simple chi-square test, to look for outliers, could be extremely deceptive in this situation, indicating significant variation when a practice was operating, on average, like all other practices. Or, put another way, 16 percent of the chi-square statistics would have been greater than 3.84 for oral examinations, giving a Type I error of 16 percent rather than the commonly accepted 5 percent level. The other Type I errors are 47 percent for fillings, 30 percent for extractions, 28 percent for root canals, and 19 percent for scalings. If these data were being used to identify outlying dentists, far more than 5 percent would be incorrectly labeled as over-providers. This discrepancy occurs because the data violate the essential property required for the chi-square test:

that the counts be independent (or, that the distribution of counts per person follow the Poisson distribution).

There is another problem with this chi-square test, which is that it ignores multiple comparisons. If a chi-square test, even using the correct 95th percentile, were performed for all 200 dentists, about 5 percent (10 practices) would be judged to have excessive utilization rates by chance alone. The probability that no dentist was classified incorrectly is $.95^{200} = .00004$. Thus, the experiment-wise Type I error is almost 1.0. This might be adjusted for by using an alpha level of $.05/200 = .00025$ instead of alpha = .05. This would require comparing the observed chi-square values to the 99.975th percentile of the distribution (about 17.17 instead of 3.84 in this situation).

Clearly, the combination of these two problems (that the chi-square statistic does not follow the chi-square distribution, and that there are multiple comparisons) will tend to yield extremely high observed chi-square values even in the null case. Users who are not aware of this problem risk labeling small areas as outliers that are not extreme at all. We have seen examples in which a large percent, or even *all* of the small areas being considered, were labeled as outliers based on this test.[19]

Analysis of Variance (F-test)

The 95th percentile of the F distribution is given in Table 4. This statistic would have resulted if we had performed an analysis of variance to detect a difference among the 200 dentists in the mean number of procedures per person. Analysis of variance requires having patient-level data, so that the variation among people can be computed. This was not available in the Washington data, but could be computed from the Pennsylvania distributions. The 95th percentile for an F distribution with 199 and infinite degrees of freedom is 1.17 if the null hypothesis is true. This is close to the simulated F values for the dental procedures (1.22, 1.13, 1.17, 1.19, 1.20), showing that analysis of variance would have been appropriate if patient-level data had been available, even though the data in Appendix I are far from normally distributed. (We used only 100 iterations, so the percentiles might be even closer than is shown.) If the data are not available at the individual level, to permit analysis of variance, it will be necessary to perform a simulation study to determine whether there is more variability than would be expected by chance alone.

Adjusting the Pennsylvania Rates

It is unfortunate that some of the average rates were so different in the Washington and Pennsylvania data sets. This suggests that some of the "significant" variation detected could have been caused not by excess variation but because the Washington data were being compared to the wrong underlying distribution. To examine this possibility we standardized the results, so that the average simulated rate would be the same as the average observed rate. Specifically, we multiplied the simulated rates by a constant factor (e.g., for oral exams 1617/1725 = 0.937 so that the simulated mean would be 1617). This has the effect of multiplying most of the values in Tables 3 and 4 by a factor less than one (0.937 for oral exams). (The EQ, CV and F values would be unchanged). The observed data would then be as extreme or even more extreme, if compared to these adjusted tables. All of the observed maxima would be larger than the adjusted 95th percentile except for root canals.

Discussion

The intent of this paper was to demonstrate that small area analysis issues were similar to those in detecting excess variation in other settings, and in particular to illustrate the simulation approach. Both the findings and the methods merit some discussion.

If we assume that the underlying distribution of procedures is about the same in the Washington and Pennsylvania data, the simulation method has demonstrated that there was excess variability for oral examinations and full mouth scaling. The other procedures also showed excess variability, although not as unequivocally.

There are some shortcomings to these results, and also to the methods that we used to obtain them. The Washington population was a fairly homogeneous group of middle-class patients. The Pennsylvania Blue Shield data were from a random sample of all insureds, who varied by social class. The differences between the two study populations may explain why the mean rates for the rehabilitative services in Table 3 were higher than those in Table 2. Also, Pennsylvania had better coverage for fillings than did the Washington contract, which may account for the discrepancy in filling rates between Tables 2 and 3.

The estimated rates from one year of Pennsylvania data were multiplied by 2 to give two-year rates for comparison with the Washington data. This is not quite appropriate, as it assumes that non-users in the first year would not have used any services in the second year. The correct multiplier is between 1.08 and 2.0 (details from authors). The "2" was chosen as an upper bound, to provide as much variability as possible. If 1.08 had been used, all of the observed maxima would have been above the 95th percentile.

No effort was made in either data set to remove people who were not covered for the entire period of time. If a person was insured for only one month of the two-year period, the rate was calculated as though the person had been at risk for the entire 24 months. This would tend to lower the rates, and would lower them more for the Washington data than for the Pennsylvania data, since the time period was longer for Washington. This may explain some of the differences in the two data sets.

We did not model one aspect of the variability—that a different number of patients might have been seen in different two-year periods. Age and sex differences were not incorporated in the simulation, as the Washington data had already been age standardized, and sex is not an important determinant of use in insured populations.[4,5] We showed elsewhere[2] that this was a reasonable way to proceed. Finally, the estimated means and percentiles are not completely accurate, as there were only 100 simulations per procedure.

Once excess variability has been established, it is appropriate to examine reasons for this variability. Grembowski, *et al.*,[10] have shown that structural features of the practice, such as its age and size, explain some of the variation in the rates.

Although not completely satisfactory, the simulation approach has provided support for the finding of excess variability among dentists. Utilization review programs operated by dental insurers are often implemented to identify dentists who have high procedure rates. The insurer may, for example, intensify review of the dentist's claims as a means of reducing the dentist's rate. Our results indicate that insurers need to determine whether excess variability exists for a given procedure before a dentist is labeled as providing "too many" services. Of equal importance, if excess variability is detected, dentists with the lowest rates may be underutilizing appropriate services.[3] Therefore, insurers should target utilization review at both ends of the rate distribution.

The methodology of this study is applicable in other situations. We used the simulation method to examine the characteristics of small area analysis statistics applied to dental practices instead of geographic areas. Dental practices have smaller numbers of patients in each "small area" and some of the procedures are applied multiple times to the same person. The distributions of root canals or scalings, which are rare, are more similar to the distribution of the number of hospital admissions for a particular diagnosis, which is often considered in small area analyses. The main results of this study have to do with the underlying distribution of services, the properties of several descriptive statistics, and various hypothesis testing procedures.

None of the distributions of services in Appendix 1 had a Poisson distribution. This is important because several approaches are now being used in the small area literature which assume that the underlying distribution is Poisson: the 1 df chi-square test and the SCV were derived under these assumptions[8] and some regression approaches model deviations from the Poisson distribution.[11,12] In all of these methods, statistically significant departures from the Poisson distribution are taken as evidence that there is unexplained variability among the small areas. However, as shown here and elsewhere,[2] the underlying distribution at the person level may not have a Poisson distribution, and finding "significant" variation is as likely to be caused by this fact as to represent excess variation among the small areas. Unless there is evidence that the underlying distribution is Poisson (e.g., that a person cannot have more than one of the procedures of interest) this is an inappropriate inference.

Tables 3 and 4 demonstrate that a considerable amount of variability can be expected among small areas by chance alone, and that it is dangerous to "eyeball" descriptive statistics as a test for excess variability. The minimum and maximum rates, and the extremal quotient, could vary considerably under the null hypothesis. The coefficient of variation and the systematic coefficient of variation also vary substantially, and tend to be larger for the less prevalent procedures (i.e., higher for root canals and scaling than for oral exams and fillings). It is common to compare CVs or SCVs across several procedures, and to claim that those with the largest coefficients represent "practitioner uncertainty." If we had done this with the data of Table 4, we would have "found" that there is less certainty about root canals and scaling than about oral exams and fillings, even though under the simulation model all dentists used procedures at exactly the same underlying rates! It is inappropriate to infer differences in "certainty" based only on CVs or SCVs if procedures have different mean rates.

Several tests of the hypothesis that there is underlying variation among the small areas have been developed. One is a 2 × k chi-square test which is appropriate if a person can have the procedure of interest at most once (e.g., death, removal of an organ). This procedure failed for the data of this study, even for procedures such as root canals, where the assumption that few people had more than one per year would have seemed "reasonable." Thus, the assumptions of this test must be examined with care.

The second chi-square test, which purports to test whether an individual dentist or small area is an "outlier" (observed significantly different from expected) was also shown to be inappropriate first, because the data do not meet the underlying assumption of a Poisson distribution, and second, because the problems of multiple comparisons are ignored. It is very important that this test be used correctly, since it singles out particular areas or dentists as outliers, and may have deleterious effects.

A third test which is sometimes proposed is a simple analysis of variance. The simulation results of Table 4 show that it would have been appropriate for these data, if data had been available at the patient level. (Such data may be difficult to obtain, as there must be a unique patient identifier to permit ascribing multiple procedures to a person.) If investigation of patient level data showed that they were inappropriate for analysis of variance, logarithmic or other transformations could be attempted.

The simulation method we have proposed has so far been used only in the null situation. The power of the various procedures to detect true underlying variation has not been studied; it should be studied. It is possible that tests based on the CV, for example, would be more powerful than other types of tests. More methods research is needed in this area.

The major lesson of this paper is that it is important in small area analysis to understand the underlying distribution of services, at the patient level, to avoid mistaking misspecification of the underlying model for statistically significant excess variability among small areas.

DISCLAIMER

Interpretations of the data are the authors' own and do not necessarily represent those of the National Center for Health Services Research and Health Care Technology Assessment, Washington Dental Service, or the Washington Education Association.

APPENDIX 1

Number of Procedures per Person in Pennsylvania Data*

Number of Procedures	Procedure Exams	Fillings	Root Canals	Full Mouth Scaling	Extractions
0.	2764	4020	6839	3722	6446
1.	2717	1239	120	43	417
2.	1349	775	16	5	88
3.	92	382	5		15
4.	39	206	3		6
5.	4	117			3
6.	14	93	1	2	10
7.		49			
8.		28	1		
9.		20			
10.		18			
11.		4			
12.	6	15			
13.		2			
14.		4			
16.		4			
18.		2			
19.		1			
21.		1			
22.		1			
24.		1			
28.		1			
29.		1			
36.		1			

*This is a sample of 12,897 people enrolled in Pennsylvania Blue Shield in 1980. Of these, 5,912 had no dental procedures in this period. These were removed from this table to match the WEA data more closely. (WEA data included only people who used one or more procedures in two years). Thus, the distributions represent the utilization of 6,985 people who had at least one dental procedure in one year. Only 3,772 of the patients had coverage for full-mouth scalings.

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Medical and Psychosocial Factors Predictive of Psychotropic Drug Use in Elderly Patients

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Abstract: The purpose of this study was to investigate medical and psychosocial factors that may be used to identify patients at risk of psychotropic drug use. Population-based surveys were completed by 278 elderly health maintenance organization (HMO) patients in August 1984. Physical and mental health status and social support were measured in the survey. Automated prescription records from the year prior to and the year after the survey were linked to data from the survey. Patients received 737 prescriptions for psychotropic drugs during the two-year period under study. Doxepin (20.2 percent), flurazepam (15.2 percent), and diazepam (14.8 percent) were dispensed most frequently. Nearly 30 percent of the patients received a prescription for at least one psychotropic drug during the two-year period, and 14 percent received at least one prescription during both years. Three significant predictors of subsequent psychotropic drug use were: prior use (odds ratio = 17.2, 95% CI = 6.25, 47.33), the number of physical impairments (OR = 1.73, 95% CI = 1.05, 2.84), and the respondent's rating on the Alameda Health Scale (OR = 1.65, 95% CI = 0.99, 2.75). Patients' self-reported mental health status and sociodemographic characteristics were not significant predictors of subsequent use. (*Am J Public Health* 1990; 80:1349–1353.)

Introduction

Patients who are 65 years or older are at risk of incurring drug-related problems, in part, because they use drugs more frequently and more intensively than younger patients.[1] They are also more likely to have one or more chronic diseases that require drug treatment, often more intensive drug treatment.[2] Psychotropic drugs are among the most frequently consumed drugs and are among the most likely to cause problems for older patients.[3] National estimates indicate that 15 percent of ambulatory males and 24 percent of females over age 65 used a psychotropic drug in 1977.[4]

Once elderly patients begin using a psychotropic drug, use may continue for an extended period of time. One study reported daily use for 12 months or more by 38 percent of antidepressant users and 15 percent of antianxiety users.[5] Another study reported that about 60 percent of elderly patients who obtained a prescription for a psychotropic drug in the preceding year also received one in the subsequent year, and the highest prevalence of extended use (i.e., over 10 years) was among patients 65 years and older.[6]

Psychotropic drug use has been repeatedly demonstrated to be associated with physical and central nervous system side effects, including reduced mental functioning,[7] addiction,[8] sleep apnea and sleep disturbances,[9] and hip fractures.[10] Correlates of psychotropic drug use include female gender,[5,11–13] psychological and physiological conditions,[14–16] and lack of a supportive social network.[17,18] No definitive and persistent associations have emerged among marital status, economic status, and class differences in the use of mood modifiers.[11]

Most studies investigating psychotropic drug use have been retrospective and have relied primarily on self-reported psychotropic drug use.[5,15,19–21] Few studies have determined patients' psychosocial characteristics during the prior year and then examined their subsequent psychotropic drug use patterns.[12,22] The purpose of the present study was to examine a group of elderly patients for predisposing medical, physical, social, and psychological factors that explain and predict their subsequent psychotropic drug use.

Methods

The Setting

The study was conducted at Group Health Cooperative (GHC) of Puget Sound, a staff model, non-profit health maintenance organization (HMO) serving approximately 340,000 members in western Washington State. A population-based survey of an age and sex-stratified random sample of 1,432 GHC adult enrollees ages 18 years and older was conducted during August 1984. Enrollees over age 65 were oversampled in order to increase the accuracy of the estimates for this age group. A description of the survey has been reported earlier and includes a comparison of the sample with the Seattle Standard Metropolitan Statistical Area (SMSA) population.[23] Completed surveys were obtained from 79 percent of the original sample. The response rate increased to 81 percent after persons were removed from the denominator who were either deceased or who were no longer enrolled in GHC at the time of the survey. The sample for the present study was a subset of 278 persons over 65 years of age who completed the survey.

In addition to their survey data, prescription records for these elderly patients were obtained from GHC's automated prescription files. Data have been collected on each prescription filled at GHC since 1977 and includes individual patient's medical history number, drug name, strength, dosage form, therapeutic category, legend status, date dispensed, and prescriber. Psychotropic drugs were an out-of-pocket expense during the study period, provided to GHC enrollees at cost. Previous studies have shown the pharmacy files are very complete and accurately reflect the actual drug use experience of GHC enrollees.[24,25]

Subsequent Psychotropic Drug Use

Subsequent psychotropic drug use was determined by an examination of the prescription records for the one-year period after the survey (September 1, 1984 to August 30, 1985). Patients were classified as users or non-users of psychotropic drugs. Users were defined as patients obtaining one or more prescriptions for an antidepressant, anxiolytic, or sedative-hypnotic drug the subsequent year. Drug prod-

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ucts were classified according to the American Medical Association Drug Evaluations.[26] A second level of use was measured by counting the number of psychotropic drug prescriptions each patient received during the year after the survey, a simple indicator of the frequency or intensity of use.

Predictor Variables

Predictor variables included the patients' psychotropic drug use during the one-year period prior to the survey, sociodemographic characteristics, social support, and medical, psychological and physical health status. Sociodemographic characteristics included the patients' marital status, living situation, age, educational attainment, income, and gender. Mental health status and social support measures were obtained primarily from the Rand Health Insurance Survey[27] and included measures of depressive symptomatology, positive outlook, and closeness/completeness of relationships.

The first physical health status measure was based on whether patients reported having suffered from each of 17 specific medical conditions or physical ailments in the preceding 12 months (1 = yes; 0 = no); the measure was the sum of all the yes responses. Patients were also asked if their physical condition sometimes interfered with (1 = yes; 0 = no): a) dressing, feeding or moving around, b) climbing stairs or getting outdoors, c) ability to work because of some illness or injury, d) changing or cutting down the kind of work, or e) cutting down or causing them to stop any other activity such as hobbies or social activities; the total number of yes responses constituted this measure. Finally, self-reported health status was measured by asking patients to compare their own health to others' their age (1 = excellent; 2 = good; 3 = fair; and 4 = poor). Self-reported health status has been shown to be related to objective measures of health status.[28–30]

The Alameda Health Status Scale[31] was also used as a composite measure of physical health status. Its categories are based on questions related to disability, impairments, chronic conditions, symptoms and energy levels, i.e. severe (1) to no disabilities/impairments and a high energy level (7). Lower scores have been shown to be associated with poorer health outcomes (e.g., higher mortality risk and long-term psychotropic drug use).[31,32]

Statistical Analysis

Psychotropic drug users and non-users were compared on each of the predictor variables to determine if there were significant bivariate differences using *chi*-square and t-tests. Logistic regression techniques were used to derive an estimate of the natural logarithm of the odds of using psychotropic drugs in the next year because the predictor variables included both categorical and continuous variables[33–35]. Proportional effects were calculated from the logistic coefficients using the mean of the dependent variable as the point of comparison. Proportional effects are interpreted as the predicted change from the point of comparison in the proportion of cases belonging to category 1 (e.g. psychotropic drug users) rather than in category 0 (e.g. non-users) for a one-unit change in the predictor variable.[36,37] A residuals regression approach was taken. Predictor variables were entered into the equation in a hierarchical fashion in order to determine change in the likelihood of psychotropic drug use over time. Prior psychotropic drug use was entered first. The sociodemographic variables were entered in the second step and the psychosocial variables were entered in the final step. The improvement of fit in the logistic models was determined by evaluating the change in the log-likelihood statistic for each step. Finally, multiple regression was used to examine the relationship between the predictor variables and the number of psychotropic prescriptions obtained during the subsequent year.

Results

The 278 patients received 737 prescriptions for psychotropic drugs during the two-year period. Anxiolytic prescriptions accounted for 40.9 percent of the prescriptions dispensed (diazepam, 15.2 percent; meprobamate, 6.9 percent; alprazolam, 4.3 percent; all others, 14.5 percent). Antidepressant prescriptions were the next most frequently dispensed (35.8 percent) and included doxepin (20.8 percent), amitryptiline (9.8 percent), trazadone (3.5 percent) and all others (1.7 percent). Finally, sedative-hypnotics accounted for 23.3 percent of the psychotropic drug prescriptions dispensed (flurazepam, 15.6 percent; triazolam, 7.2 percent; pentobarbital, 0.5 percent).

Over 30 percent of the patients obtained at least one prescription for a psychotropic drug during the two-year period studied (Table 1). Nearly 23 percent of the patients had between one and 10 prescriptions filled, whereas over 8 percent of them received 11 or more prescriptions (range 0 to 39 prescriptions per patient). When only patients obtaining one or more psychotropic prescriptions were examined, they averaged nearly nine prescriptions per patient and over 60 percent of the patients that obtained a psychotropic prescription during the first year obtained another during the subsequent year. Nearly 53 percent of the patients who obtained a single prescription during the two-year study received an anxiolytic and 33 percent received a sedative-hypnotic. On the other end of the continuum, one patient received 39 psychotropic prescriptions (34 antidepressant prescriptions

TABLE 1—Frequency and Intensity of Psychotropic Drug Use

Prescriptions/Patient			Quantity/Prescription		
Prescriptions	No.	Percent	Pills	No.	Percent
None	193	69.4	10 or less	52	7.1
1–5	50	18.0	11–19	39	5.3
6–10	12	4.3	20–29	103	14.0
11–20	10	3.6	30–49	188	25.6
21–30	10	3.6	50–99	228	30.9
30 or more	3	1.2	100–199	90	12.2
			200 or more	37	5.0
TOTAL	278	100.0	TOTAL	737	100.0

and five anxiolytic prescriptions) and another patient received 36 prescriptions (all for anxiolytics).

Table 2 shows the results of the comparisons on each of the sociodemographic and health status predictor variables during the subsequent year between users and non-users of psychotropic drugs. The average user was nearly two years younger than the average nonuser. Psychotropic users reported that their physical condition interfered with more of their daily activities, had more self-reported medical conditions, had a less positive outlook on life, and, according to their own reports, were in somewhat poorer health. It is interesting to note that while self-reported health status of users was significantly lower than that of non-users, it was still in the "good" to "fair" range.

Multiple logistic regression results showed that prior drug use was the most important predictor of subsequent use (Table 3). A patient using a psychotropic drug during the year prior to the survey was 17.2 times (95% CI = 6.25 to 47.33) more likely to be a user during the year following the survey when compared to a non-user in the year before the survey. Stated another way, psychotropic drug use during the year before the survey increased the probability of being a user in the year after the survey to over 80 percent. The sociodemographic variables did not contribute to the prediction of subsequent use with the prior drug use variable in the model. For each additional daily activity interfered with, or for each additional unit increase on the Alameda Health Index, the probability of using a psychotropic drug increased 9.9 and 8.9 percent, respectively. The two mental health status variables (depressive symptomatology and positive outlook) were not significant when use in the prior year and physical health status were simultaneously controlled. Addition of the psychosocial and physical health status variables as a group did not significantly improve the fit of the equation over just prior use alone. In a separate analysis, the sociodemographic, psychosocial and physical health status measures were entered first into the equation (as a proxy for psychosocial and physical health status *prior* to psychotropic drug use in the year before the survey). Adding these variables first into the equation did not improve the fit of the model over the constant alone (change in *chi*-square goodness-of-fit = 5.17, 11 df). When drug use in the prior period was next added to the equation, the fit of the model improved (change in *chi*-square goodness-of-fit = 35.54, 1 df).

The only significant predictor of the number of psychotropic prescriptions obtained during the year following the survey was the number of psychotropic prescriptions obtained in the year before the survey (Table 4). The large positive relationship between the prior year's use and the subsequent year's use confirms that patients obtaining a larger number of prescriptions in the prior year are more likely to obtain a relatively larger number of prescriptions in the subsequent year.

Discussion

Our results show that elderly patients at the most risk of receiving a prescription for an anxiolytic, antidepressant or sedative-hypnotic drug in the following year were those who had used one in the previous year. Patients at greater risk were also more likely to be in somewhat poorer physical health and to have their daily activities interfered with to a somewhat greater extent. Patients' sociodemographic characteristics and measures of their mental health status were not significant predictors of subsequent psychotropic use. The frequency or intensity of subsequent drug use was also closely associated with the previous year's experience.

TABLE 2—Comparisons of Users and Non-users of Psychotropic Drugs in the Year after the Survey

Variables	Users (N = 59)	Non-users (N = 219)	Test Statistic	Difference	95% CI
Age	72.9	74.9	2.17[a]	1.98	0.09, 3.87
Percent female	66.1	54.8	1.98[b]	0.16	
Percent married	59.3	64.8	0.39[b]	0.52	
Percent living alone	27.1	28.3	0.00[b]	0.99	
Education (percent)			2.83[c]		
<8th grade	15.8	11.7			
9–11th grade	24.6	20.4			
High school graduate	24.6	26.3			
13–15 years	21.1	23.0			
College graduate	8.8	7.5			
Graduate school	5.3	11.3			
Income (percent)			3.12[c]		
<$10,000	26.5	23.5			
$10–$15,000	32.7	25.7			
$15–$25,000	22.4	29.9			
$25–$35,000	14.3	11.8			
$35–$50,000	2.0	5.9			
$50,000+	2.0	3.2			
Number of daily activities interfered with	1.04	0.61	2.09[a]	0.43	−0.03, 0.89
Number of medical conditions	4.32	3.42	2.13[a]	0.90	0.03, 1.79
Alameda scale	3.01	3.39	1.55[a]	0.38	−0.13, 0.88
Emotional ties	9.57	9.69	0.32[a]	0.12	−0.65, 0.99
Positive effect	43.89	46.77	1.91[a]	2.88	−0.21, 5.99
Depressive symptomatology	7.18	6.76	0.85[a]	0.42	−0.41,1.25
Self-reported health status	2.23	1.95	2.56[a]	0.28	0.02, 0.52

[a] Independent t-test
[b] *chi*-square (df = 2)
[c] *chi*-square (df = 5)

TABLE 3—Effects of Prior Use, Sociodemographic, and Psychosocial Characteristics on Subsequent Psychotropic Drug Use

Independent Variables	Step 1 β	Step 2 β	Step 3 β	% Predicted Proportional Change (Step 3)	Odds Ratio	95% Confidence Interval
Prior use	2.68*	2.66*	2.85*	61.1	17.2	6.25, 47.33
Sociodemographics						
Age	—	.033	.035	0.1	1.04	0.94, 1.14
Sex (female = 1)	—	.098	.008	0.2	1.01	0.31, 3.33
Married	—	.089	.093	1.5	1.10	0.14, 8.87
Living alone	—	.074	.100	1.6	1.11	0.14, 8.67
Physical and Psychosocial Characteristics						
Self-reported health status	—	—	.014	0.2	1.01	0.43, 2.41
Depressive symptomatology	—	—	.084	1.4	1.09	0.89, 1.33
Positive outlook	—	—	.008	0.1	1.01	0.92, 1.10
Emotional ties	—	—	.025	0.4	1.03	0.79, 1.33
Alameda scale	—	—	.501*	8.9	1.65	0.99, 2.75
No. of impairments	—	—	.549*	9.9	1.73	1.05, 2.84
No. of conditions	—	—	.087	1.4	1.09	0.84 ,1.42
Mean, Dependent	.1905	.1905	.1905			
Intercept	2.32	4.64	8.71			
chi-square goodness of fit (intercept only = 163.03)	126.87	126.18	119.32			
chi-square compared to intercept only:	36.16/1 df	36.85/5 df	43.71/12 df			
Change:	36.16/1 df	0.59/4 df	6.96/7 df			
p-value of change	p < .005	p = n.s.	p = n.s.			

*p < .05 coefficient/s.e. coeff which approximates t-distribution.

TABLE 4—Stepwise Multiple Regression of Predictor Variables on the Number of Prescriptions Obtained during the Subsequent Year

	Step 1 Beta	Step 2 Beta	Step 3* Beta	Standard Error B
Prior Use	0.67	0.67	0.64	0.05
Sociodemographics				
Age	—	0.08	0.07	0.02
Sex	—	0.01	0.01	0.22
Married	—	0.04	0.06	0.37
Living alone	—	0.06	0.06	0.38
Physical and Psychosocial Characteristics				
Self-reported health status	—	—	0.06	0.16
Depressive symptomatology	—	—	0.01	0.04
Positive outlook	—	—	−0.06	0.02
Emotional ties	—	—	0.03	0.06
Alameda scale	—	—	0.02	0.09
No. of impairments	—	—	0.05	0.11
No. of conditions	—	—	0.01	0.05
Adjusted R^2	0.45	0.45	0.45	
N	278	278	278	
F-ratio	223.0	45.4	19.3	

*Residuals analysis identified three outliers. The adjusted R^2 increased to 0.57 when the three outliers were eliminated.

Our finding that psychotropic drug use is independent of patients' physical and mental health status is consistent with the work of others.[38] There was also evidence of an association between physical disorders and the likelihood of any use, but not with the number of psychotropic prescriptions obtained during the subsequent year.

One reason for the predominant role of prior drug use as a predictor of later use, especially when compared to the findings of previous studies, may relate to the differences in data collection methods. In most of the previously cited retrospective studies, patients were asked to recall their psychotropic drug use during a period prior to collection of the psychosocial information. In terms of cause and effect, this more closely represents the notion that the psychotropic drug caused the psychosocial condition, not vice versa. In this study, we collected psychotropic drug use from prescription records before and after assessment of patients' sociodemographic and psychosocial characteristics.

Limitations to the study design exist that may constrain interpretation of the data. Psychotropic drug users were identified exclusively by automated pharmacy records. The GHC pharmacy data base provides a reasonably complete record of receipt of drugs; however, patients' actual consumption was unconfirmed. In addition, patients' sociodemographic characteristics and physical and mental health status data were obtained exclusively by self-reports.

Further work is needed to examine predictors of individual psychotropic drug categories. Small numbers of users prohibited such an analysis in this study. The sample size also influenced the time interval chosen for the present study. It would be beneficial to examine whether patient characteristics and mental health status were more important if the time interval were shorter than one year.

Another limitation of these data is the nature of the sample. The statistical findings are governed not only by its size, but also by the distribution of the variables within the sample. The distribution may differ in another sample. Therefore, caution should be used when generalizing our findings beyond the population of elderly patients from where our random sample was drawn.

Psychotropic drugs are often legitimately used for short-term treatment of physical disorders associated with normal aging; however, long-term use has been questioned.[39] Long-term psychotropic drug taking may be a learned behavior directed at helping elderly patients deal with chronic physical and emotional problems or reducing anxiety attacks and physical symptoms associated with withdrawal syndrome.[39–41] Prescription for a psychotropic drug may also serve to diminish physicians' frustration in treating the many ambiguous conditions associated with normal aging and to appear supportive to the patient.[38,42–43] Examination of the specific reasons for prescribing psychotropic drugs was beyond the scope of this study. However, we did document a pattern of continued use among a small portion of the elderly population that could not be explained or predicted by the level of patients' sociodemographic, mental and physical health status, and social support.

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The Association between Type A Behavior and Change in Coronary Risk Factors among Young Adults

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Abstract: The association of Type A/B behavior pattern and changes in blood pressure, total serum cholesterol, serum triglyceride, body mass, and smoking was estimated in a cohort of 375 young Black and White men and women from a rural county in Central Kentucky between 1978–79 and 1985–88. Type A participants experienced significant increases in systolic (2.90 ± 1.29 mmHg) and diastolic (3.80 ± 1.17 mmHg) blood pressure and in cigarette smoking (3.26 ± 0.89 cigarettes per day) over the eight-year follow-up period, but Type B participants experienced no change. Type A and B individuals showed similar changes in total serum cholesterol, serum triglyceride, or body mass. Differences between behavioral types in blood pressure were present for women but not men, and for Blacks but not for Whites. These findings suggest a possible significance of the Type A pattern for the development of cardiovascular risk of young adults. (*Am J Public Health* 1990; 80:1354–1357.)

Introduction

Cardiovascular epidemiology has established blood pressure, serum cholesterol, and smoking as the most powerful and consistent predictors of coronary risk in middle age and older adults. Studies of children suggest that coronary risk factors are already present at early ages,[1,2] but relatively little epidemiologic data exist on the coronary risk status of young adults, especially those who have recently completed physical growth as they begin to enter typical adult roles in this society,[3] a phase characterized by changes in lifestyle and responsibility. The student becomes a worker, the single person a spouse and parent, with changes in eating, drinking, sleeping, and exercise.

The style of coping with these challenges may have a bearing on level of life stress and, through it, on markers of coronary risk. Type A, coronary-prone behavior pattern is believed to relate to risk for coronary heart disease.[4] We followed a sample of men and women from small town and rural backgrounds from an average age of 21 years to an average age of 29 to determine whether Type A/B behavior pattern is associated with change in coronary risk factors. Previously, we have shown clustering of risk factors in individuals in this population.[5]

Methods

The sample for the present study was derived from a 15-year longitudinal cohort study of young adults in a rural county of the Central Kentucky Bluegrass. In 1973, all high school students of Bourbon County, Kentucky (N = 1,142) were assessed for their blood pressures, body weights, and heights, as well as a few other demographic characteristics. Students' ages ranged from 14 to 19 years.

In 1978–79, a sample (N = 472) was drawn from the original study population to include the 20 percent of young adults who had the highest and lowest systolic blood pressure readings when they were teenagers in 1973. Because some of the subjects had not yet achieved adult growth at baseline, all of the subjects who were less than 16 years old in 1973 were also contacted for re-examination in 1978. Of the remaining group, 15 percent were selected at random for follow-up. Further description of this sample has been published previously.[5–7]

In 1985–88, the attempt was made to contact the entire population studied at baseline in 1973. Of the original population, 253 were lost to the study; 219 could not be located; 19 refused; 15 had died. Follow-up data were successfully gathered on 891 subjects, or 78 percent of the original pool.

The sample for the present study (N = 375) includes subjects for whom data are available both from the 1978 study and from the 1985–88 population follow-up. Subjects with very high or very low systolic blood pressure measurements when they were teenagers are overrepresented in this group, as are younger participants. As seen in Table 1, the typical 1985–88 participant was a White, married, full-time employed, high school graduate born in 1958. About half of the study group was male.

At both 1978–79 and 1985–88 data collections, participants were located by searches of local public documents, contacts with relatives and friends of the participants, and by placing classified advertisements in local and regional newspapers. Measurements were usually made at the project office in Bourbon County. For participants not willing to travel to the project office, home visits were performed by project staff throughout the state of Kentucky. For two out-of-state participants, Physical Measurements, Inc was hired to provide home visits at which a blood sample, blood pressure, and other anthropometric measurements were obtained according to protocol. Questionnaires were also completed by these two participants. Blood pressure, anthropometric, and medical history data were collected at both waves. The Type A measure (the Jenkins Activity Survey, Form C)[8] was administered only in 1985–88. Type A behavior was measured with the JAS Type A subscale comprising 21 items from the longer instrument. Each response was assigned a weight and summed according to the method of Jenkins.[8] Blood lipid analyses and other cardiovascular assessments[7] are available on a subset of the participants (N = 98).

Quetelet index[9]: Q = weight in kilograms/(height in meters)^2 was calculated. Blood pressure was measured in the right arm after the subject was seated for at least five minutes.

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TABLE 1—Distributions of Selected Demographic Characteristics of Participants, Bourbon County, Kentucky, 1978–79 and 1985–88 (N = 375)*

	1978-79		1985–88	
Age (years)	20.7 ± 1.8		28.6 ± 1.4	
Sex	N	(%)		
Male	200	(53.3)		
Female	175	(46.7)		
Marital Status			N	(%)
Single	251	(67.1)	101	(26.9)
Married	116	(31.0)	227	(60.5)
Separated	4	(1.1)	8	(2.1)
Divorced	3	(0.8)	39	(10.4)
Widowed	0	(0.0)	0	(0.0)
Employment				
Fulltime	90	(24.0)	283	(75.5)
Parttime	227	(60.5)	25	(6.7)
Not employed	58	(15.5)	67	(17.9)
Education				
Some High School	42	(11.2)	42	(11.2)
High School Diploma	243	(64.8)	232	(61.9)
Some College	83	(22.1)	53	(14.1)
College Diploma	7	(1.9)	48	(12.8)
Race				
White	301	(80.3)		
Black	74	(19.7)		

*375 is the total number of participants. Smaller numbers are due to missing information on some variables.

Three measurements by a trained observer were taken over a 10–15 minute period using a standard mercury manometer and cuff calibrated for arm circumference; these readings were averaged. Blood samples were analyzed for total serum cholesterol and serum triglyceride values by the autoanalyzer method described in the Lipid Research Clinics Manual of Laboratory Operations.[10]

We studied systolic and diastolic blood pressure, serum cholesterol, and serum triglyceride levels, body mass, and smoking behavior as indicators of risk. Change in risk status was measured as the difference in scores from 1978–79 to 1985–88 on each of these variables. Using change scores for each of the risk factors as outcomes, we calculated a three-way factorial model for all risk factors except smoking change, with behavior pattern, race and sex as the explanatory variables. Change in number of cigarettes smoked was treated as a categorical variable for log-linear modeling and contingency table analysis. Race, sex and body mass were included as potential control variables.[12] Relations between behavior pattern and risk variables were affected by race and sex, but not by change in body mass (data available on request) so change in body mass was removed from further analyses.

Results

Coronary risk factor means and standard deviations at the two time periods are shown in Table 2.

The three-way factorial analysis for change in systolic blood pressure revealed that only the main effect of behavior pattern accounted for significant variance in change in systolic blood pressure between 1978–79 and 1985–88 assessments. As shown in Table 3, Type A subjects experienced a mean 2.90 ± 1.29 (standard error) mmHg change, whereas Type B subjects showed a mean −1.47 ± 1.22 mmHg change in systolic blood pressure. The Type A subjects experienced greater increases in systolic blood pressure than Type B subjects in the entire sample (p = 0.01), in women (p = 0.02) but not in men (p = 0.23), and in Blacks (p = 0.05) but not in Whites (p = 0.12).

TABLE 2—Coronary Risk Factors in Participants, Means and Standard Deviations, Bourbon County, Kentucky, 1978–79 and 1985–88

	N	1978–79	1985–88
		Means SD	Means SD
Systolic Blood Pressure	375[+]	120.27 ± 14.90	120.69 ± 16.07
Diastolic Blood Pressure	375	73.08 ± 11.35	75.28 ± 11.88
Serum Cholesterol	98	167.21 ± 37.32	204.19 ± 43.18
Serum Triglyceride	98	107.53 ± 77.81	168.36 ± 99.65
Body Mass Index	371	24.17 ± 6.48	26.62 ± 5.81
JAS Type A Score	375		211.28 ± 72.82
Cigarettes/Day	369	7.24 ± 10.99	9.18 ± 13.39

+ Fewer than 375 cases on a variable are due to missing information (except for the serum cholesterol and serum triglyceride variables in which sample design called for fewer cases).

In the three-way factorial analysis for change in diastolic blood pressure, none of the explanatory variables or their interactions were statistically significant, but Type A individuals rose an average 3.80 ± 1.17 mmHg, whereas Type B individuals rose 0.50 ± 1.11 mmHg in diastolic blood pressure (Table 4). The Type A subjects experienced somewhat larger increase in diastolic blood pressure than Type B subjects in each contrast especially among Blacks and women.

Factorial analysis of changes in total serum cholesterol, serum triglyceride, and body mass values showed no statistically significant main or interaction effects on change in any of these three risk variables.

Log-linear modeling analysis was performed with smoking, the outcome, divided into participants who decreased, remained unchanged and increased cigarette consumption from 1978–79 to 1985–88, and with behavior pattern, sex and race the predictors. Only behavior pattern was significantly associated with smoking change. Table 5 shows that Type As increased smoking more than type Bs, and that Type Bs held their smoking at the same level to a greater extent than Type As. Log-linear analysis found that the behavior pattern/smoking change association was statistically significant in Black females but not in the other three race/sex groups.

Discussion

In 1981, an authoritative panel examined the available evidence and judged the Type A behavior pattern to be a risk factor for coronary disease, comparable in impact to smoking, elevated cholesterol, and elevated blood pressure.[4] Subsequent results call that conclusion into question.[13–15] Recent research suggests that underlying hostility is the aspect of Type A behavior that is most strongly predictive of coronary heart disease endpoints.[16] A reanalysis of the Multiple Risk Factor Intervention Trial (MRFIT) data, one of the major recent studies failing to find a Type A/coronary disease association, has found indicators of hostility to significantly predict coronary disease incidence.[17]

Appropriate measurement of Type A behavior is uncertain,[18] but at best is only modestly associated with other coronary risk factors,[19,20] and has been seen as an independent risk factor. This study suggests that, in young adults, Type A behavior pattern is associated with more rapid increases in two well-accepted coronary risk factors. Type A behavior may operate differently at different ages to produce

TABLE 3—Mean Change (± standard error) in Systolic Blood Pressure for Sex/Race/Behavior-Pattern Groups*

	Males		Females		Both Sexes		
	Type A	Type B	Type A	Type B	Type A	Type B	A vs. B (p)
White	1.17	−0.66	1.72	−1.40	1.44	−1.03	0.12
	(1.38)	(1.65)	(1.68)	(1.58)	(1.09)	(1.14)	
Black	4.93	0.73	3.79	−4.56	4.36	−1.91	0.05
	(3.49)	(2.88)	(3.10)	(3.19)	(2.34)	(2.15)	
Both Races	3.05	0.04	2.76	−2.98	2.90	−1.47	
	(1.88)	(1.66)	(1.76)	(1.78)	(1.29)	(1.22)	
A vs B (p-value)	0.23		0.02				0.01

*Three-way analysis of variance found a significant ($p < .05$) main effect for Type A/B behavior pattern but no other main or interaction effects.

TABLE 4—Mean Change (± standard error) in Diastolic Blood Pressure for Sex/Race/Behavior-Pattern Groups*

	Males		Females		Both Sexes		
	Type A	Type B	Type A	Type B	Type A	Type B	A vs. B (p)
White	2.16	1.76	3.08	2.27	2.62	2.02	0.68
	(1.26)	(1.50)	(1.53)	(1.44)	(0.99)	(1.04)	
Black	4.40	0.00	5.58	−2.00	4.99	−1.00	0.04
	(3.18)	(2.62)	(2.82)	(2.90)	(2.13)	(1.96)	
Both Races	3.28	0.88	4.33	0.14	3.80	0.50	
	(1.71)	(1.51)	(1.60)	(1.62)	(1.17)	(1.11)	
A vs B (p-value)	0.29		0.07				0.04

*Three-way analysis of variance found no significant main or interaction effects.

TABLE 5—Contingency Table Analysis (chi-square) of the Association between Behavior Pattern and Change in Smoking Behavior, 1978–79 to 1985–88 (N = 369)

	Behavior Pattern			
	Type B		Type A	
Smoking Change	No.	%	No.	%
Decreased	21	11.8	28	14.7
Stayed Same	121	68.0	107	56.0
Increased	36	20.2	56	29.3
Total	178	100.0	191	100.0

chi-square = 5.76; d.f. = 2; p = 0.056

eventual coronary disease endpoints. In young adulthood, Type A might promote exacerbation of two coronary disease risk factors and thereby support early atherogenesis. In these younger adults, the Type A pattern may reinforce the smoking habit by motivating individuals toward greater cigarette consumption. The Type A pattern may also stimulate more rapid escalation of blood pressure in ways analogous to those demonstrated in stress laboratory research on Type A responsiveness to hostile challenges.[18] Type A behavior may become less strongly associated with these risk factors in middle age as life situations become more predictable and manageable.

Whether a Type A measure taken in 1985–88 reflects status in 1978–9 might be questioned, but Type A measures have been shown to be stable in young adults and in children.[21–23] At the least, these data suggest that those who measure as Type A in their late 20s are more likely to have experienced a steeper increase in blood pressure and smoking during the preceding decade than their Type B counterparts.

In the study of "Framingham offspring,"[3] their lifestyle, and changes in serum cholesterol, lipoproteins and blood pressure, no association was found between Framingham Type A measures (total scale, trait subscale, and job-related subscale) and changes in systolic or diastolic blood pressure, total, high density lipoprotein, low density lipoprotein, very low density lipoprotein cholesterols, and the ratio of total to high density lipoprotein cholesterols. However, in men only, the Framingham Type A trait subscale was independently predictive of the ratio of total to high density lipoprotein cholesterols by regression analysis. This significant finding could not be examined in the present study because of our lack of lipoprotein data. We are also unable to compare our body mass, serum triglyceride, and smoking results to those of the Framingham offspring study as the latter did not report Type A by body mass, serum triglyceride, and smoking associations.

Neither the design nor the data of the present study permits firm explanations for the stronger association of Type A with blood pressure and smoking changes in women and Blacks than in men and Whites. One possible explanation is that Type A women and Blacks, because of social discrimination, find themselves more consistently engaging in the Type A coping responses to challenges.

In summary, this nine-year cohort study uncovered evidence that individuals with the JAS-measured Type A behavior pattern were likely to experience a steeper increase in blood pressure and rate of cigarette smoking during their third decade of life than their Type B counterparts. These results were found in analysis of the whole study group, but appeared stronger in women and Blacks for blood pressure, and in Black women for smoking. In light of earlier findings of no relationships between Type A behavior and coronary disease risk factors, these results suggest that age differences may affect the strength of those associations.

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Behavioral Covariates of Waist-to-Hip Ratio in Rancho Bernardo

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Abstract: We examined lifestyle and dietary habits in 685 men and 943 women (mean age 67 years) who completed an interview, examination, and food frequency questionnaire in 1984–87. Waist-to-hip ratio increased with age and body mass index in both men and women. In multiple regression, waist-to-hip ratio was independently associated with smoking, alcohol consumption, and exercise in men, and with smoking and alcohol consumption in women. The data suggest that waist-to-hip ratio is affected, at least in part, by behavioral, and potentially modifiable, factors. (*Am J Public Health* 1990; 80:1358–1362.)

Introduction

Male pattern abdominal or truncal obesity, as measured by either waist-to-hip girth ratio, or ratio of subscapular to triceps skin-fold, has been implicated in the development of ischemic heart disease,[1–3] stroke,[3,4] diabetes,[5,6] hypertension,[7–9] and all-cause mortality[2] in both men and women. The association is independent of overall adiposity. In fact, in at least one study, those at highest risk for subsequent ischemic heart disease and death were the leanest subjects, with the greatest waist-to-hip girth ratios.[2]

The etiology of abdominal obesity is not clear. Studies by Bouchard[10] have shown that heredity accounts for only 20–25 percent of the variance in waist-to-hip ratio in the general population. Shimokata[11] and we[12] recently reported that cigarette smoking is related to abdominal obesity independently of age and body mass index. However, since smoking is highly correlated with other health behaviors, such as diet, alcohol consumption, and physical activity, it was unclear whether the increased abdominal adiposity seen in smokers is due to smoking per se, or is a consequence of other, associated behaviors.

In this report we have attempted to determine whether other behaviors, specifically alcohol consumption, physical activity, and diet, also contribute to the waist-to-hip ratio in a defined population.

Methods

The population for this study consisted of older adult residents of Rancho Bernardo, a geographically defined community of Caucasian upper middle-class in Southern California. These subjects were first surveyed in 1972–74 as part of the Lipid Research Clinics Prevalence Study. As previously reported,[13] the initial response rate was 82 percent. In 1984–87, 81 percent of surviving men and women participated in a follow-up evaluation by telephone and/or clinic visit. The 76 percent who were seen in clinic form the basis of these analyses.

Smoking and alcohol use were determined by standardized, self-administered questionnaires which asked subjects if they had consumed any alcohol in the past year, the frequency of their consumption, and the specific forms of alcohol consumed in an average week and in the week prior to the survey. Grams of alcohol for the week prior to the survey and for an average week were computed by means of an algorithm.[14] For smoking, participants were asked if they had ever smoked cigarettes, whether they currently smoked, the usual number of cigarettes smoked per day, and number of years smoked. Exercise was assessed by asking participants if they regularly exercised three times per week. Diet was assessed in a subsample of 737 participants with the Harvard-Willett semi-quantitative food frequency questionnaire.[15]

In clinic, a nurse measured height and weight with subjects in light clothing, without shoes. Body mass index (weight in kg/height in m^2) was calculated as an estimate of obesity. Waist and hip girths were measured over light, single thickness clothing with participants standing in an erect position with feet together. Participants were asked to bend forward and return to an erect posture. Waist girth was measured in cms at the bending point. Hip girth was measured in cms at the iliac crest. A 75 gram oral glucose tolerance test was administered in the morning after a requested 12-hour fast; diabetes was defined by World Health Organization (WHO) criteria.[16]

Among the 841 men and 1,117 women ages 50–79 who participated, 125 men and 114 women had non-insulin dependent diabetes by history or glucose tolerance test. Non-insulin dependent diabetes, which is strongly genetically determined,[17] and related to waist-to-hip-ratio was excluded from the present analyses in order to emphasize attributes which could be modified by behavior. Subjects with incomplete diabetes data ($N = 84$) were likewise excluded. These analyses, therefore, are based on 685 men and 943 women without diabetes. There were no other exclusions for disease, but all subjects were healthy enough to come to the clinic.

Data were analyzed using Statistical Analysis Systems (Cary, NC). Groups were compared using analysis of variance; age and age and body mass index-adjusted values for waist-to-hip ratio were compared by analysis of covariance. Pearson correlation coefficients were used to assess the correlation of waist-to-hip ratio with the dietary variables. Waist-to-hip ratio was sufficiently normally distributed not to require transformation. Multiple linear regression was used to determine the independent contributions of the behavioral variables to waist-to-hip ratio. All probabilities reported are for two-tailed tests.

Results

The mean age of both men and women was 67 years (Table 1). Men were heavier and had a higher waist-to-hip ratio than women. BMI decreased with age in men ($r = -.19$, $p < .001$) but not in women. Waist-to-hip ratio correlated with BMI in both men and women, ($r = .40$; $r = .31$, respectively).

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TABLE 1—Clinical and Behavioral Characteristics of Men and Women Ages 50–79 in Rancho Bernardo

	Men N = 685	Women N = 943
Age (mean years)	67.5 (8.3)	67.6 (8.2)
BMI (mean kg/m^2)	26.2 (3.3)	24.4 (3.7)
Waist-to-Hip Ratio	0.915 (.050)	0.789 (.062)
Smokers		
% Never	28	47
% Former	60	37
% Current	12	16
Cigarettes per day* (mean)		
Former	26 (16)	16 (15)
Current	24 (17)	16 (13)
% Drinkers	75	65
Alcohol past week** (mean gms)	142 (126)	102 (92)
Alcohol average week** (mean gms)	152 (123)	111 (88)
% Exercisers	87	80

Values are mean ± SD.
*among smokers
**among drinkers

When adjusted for BMI, waist-to-hip ratio increased with age in both men and women (r = .11, r = .23, respectively).

Almost two-thirds of the men and over one-third of the women had smoked in the past, but only 12 percent of the men and 16 percent of the women were current smokers. Over two-thirds of men and women reported drinking at least one or two drinks per month and most reported regular physical activity. Among users, men smoked more cigarettes and drank more alcohol than women.

Waist-to-hip ratio was higher in men and women who had ever smoked compared to those who had never smoked, and was higher in the upper quartile of drinkers (>9 oz/wk for men; >7 oz/wk for women) compared to all others. Waist-to-hip ratio was lower in men and women who reported regular exercise compared to those who did not (Table 2).

Waist-to-hip ratio, adjusted for age and BMI, was lowest in never smokers, intermediate in former smokers, and highest in current heavy smokers (Figure 1). The magnitude of the difference in waist-to-hip ratio between never smokers and current heavy smokers was as large in the women as in the men, despite the fact that within each category the women smoked fewer cigarettes per day.

Figure 2 shows mean waist-to-hip ratios by five drinking categories based on alcohol consumption in the week prior to the clinic visit week. Similar results (not shown) were found based on alcohol consumption in an average week. In both men and women, the heaviest drinkers had the highest waist-to-hip ratios.

Correlations between a number of dietary variables from the Harvard-Willett food-frequency questionnaire and waist-to-hip ratio were examined in 335 men and 402 women, shown in Table 3. Among the dietary variables, percent calories from alcohol was positively correlated with waist-to-hip ratio in men and women, as was absolute alcohol intake (data not shown). The only other dietary variable significantly correlated with waist-to-hip ratio was percent calories from carbohydrate (exclusive of alcohol) which was inversely correlated with waist-to-hip ratio in men.

Cigarettes per day for former and current smokers, alcohol as a continuous variable, and exercise were entered into a multiple linear regression model with age and BMI. There was no significant interaction of smoking with alcohol intake in the model. As shown in Table 4, in both sexes, age, BMI, alcohol intake, and smoking were independently associated with waist-to-hip ratio. Exercise was independently associated with waist-to-hip ratio in men but not in women. Overall, these attributes explained 21 percent of the variance in waist-to-hip ratio in men and 16 percent in women.

Discussion

The study confirms observations by Shimokata,[11] and extends earlier observations in this population[12] by demonstrating that effects of smoking and drinking on waist-to-hip ratio are independent and additive.

The fact that the increase in waist-to-hip ratio associated with drinking was accounted for by the heaviest drinkers is consistent with one possible mechanism, an increase in adrenal cortisol production which has been reported in alcoholics and has been termed "alcohol-induced pseudo-Cushing's syndrome."[18,19] It is well-known that central obesity is a prominent feature of Cushing's syndrome.[20] Although pseudo-Cushing's is apparently a relatively uncommon complication of alcoholism,[21] it is possible that a lesser degree of abnormality in cortisol secretion associated with heavy drinking could affect waist-to-hip ratio in the absence of clinical Cushing's syndrome.

The association of smoking with increased waist-to-hip ratio in women may be explained by the fact that women smokers have increased levels of adrenal androgens[22,23]

TABLE 2—Mean Waist-to-Hip Ratio by Presence or Absence Behavioral Variables

	Men			Women		
	N = 685			N = 943		
Behavior	Presence	Absence	Difference (95% CI)	Presence	Absence	Difference (95% CI)
Smoker (ever)						
Unadjusted	.918	.906	.012 (.004, .020)	.794	.784	.010 (.002, .018)
Age adjusted	.918	.906	.012 (.004, .020)	.794	.784	.010 (.003, .018)
Age and BMI adjusted	.918	.906	.012 (.004, .019)	.795	.783	.012 (.004, .019)
Drinker (upper quartile vs all others)						
Unadjusted	.928	.912	.016 (.007, .026)	.804	.786	.018 (.007, .028)
Age adjusted	.928	.912	.016 (.007, .026)	.803	.787	.016 (.006, .026)
Age and BMI adjusted	.929	.912	.017 (.008, .026)	.803	.787	.016 (.007, .026)
Exerciser (regular)						
Unadjusted	.912	.933	−.021 (−.010, −.032)	.787	.797	−.010 (−.001, −.020)
Age adjusted	.912	.934	−.022 (−.010, −.033)	.787	.797	−.010 (−.001, −.020)
Age and BMI adjusted	.913	.930	−.017 (−.007, −.027)	.789	.797	−.004 (.005, −.013)

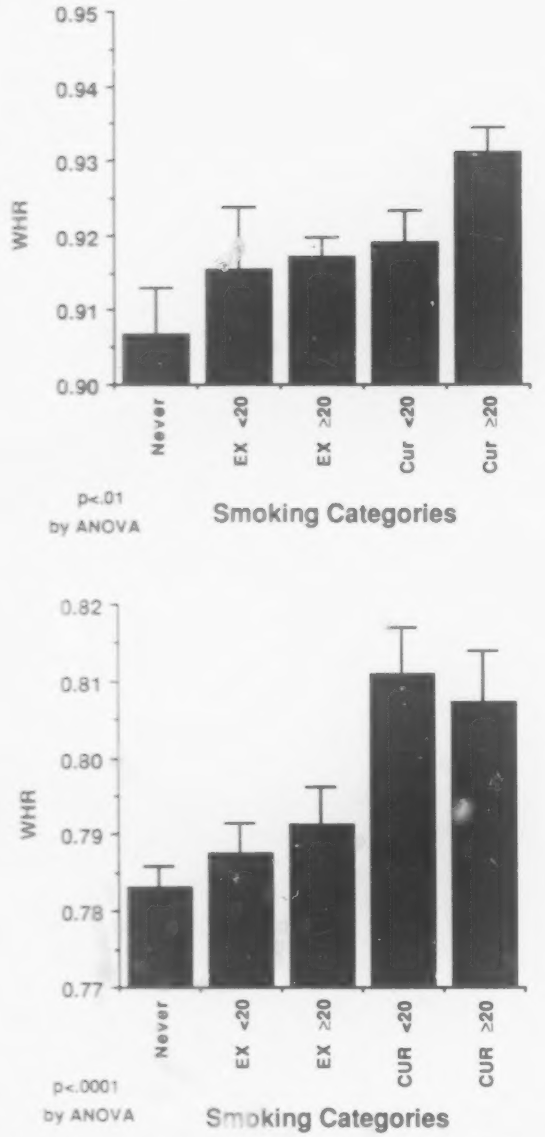

FIGURE 1—Mean Waist-to-hip Ratios Adjusted for Age and BMI by Category of Smoking: Never Smokers, Exsmokers of <20 Cigarettes per Day, Exsmokers of ≥20 Cigarettes per Day, Current Smokers of <20 and ≥20 Cigarettes per Day (Men are shown in the upper panel; women in the lower.)

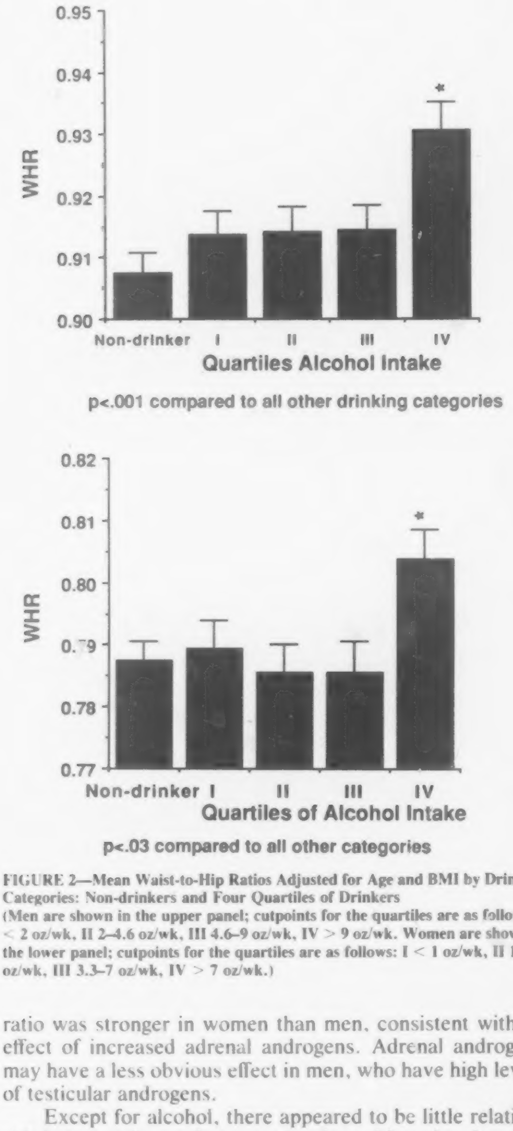

FIGURE 2—Mean Waist-to-Hip Ratios Adjusted for Age and BMI by Drinking Categories: Non-drinkers and Four Quartiles of Drinkers
(Men are shown in the upper panel; cutpoints for the quartiles are as follows: I < 2 oz/wk, II 2–4.6 oz/wk, III 4.6–9 oz/wk, IV > 9 oz/wk. Women are shown in the lower panel; cutpoints for the quartiles are as follows: I < 1 oz/wk, II 1–3.3 oz/wk, III 3.3–7 oz/wk, IV > 7 oz/wk.)

which have been shown to be associated with upper body obesity.[24] Increased cortisol levels in postmenopausal women smokers[23] also may contribute to increased abdominal obesity. In men, smoking is associated with increased adrenal androgens and estrogen, however, there are few data on the association of sex hormones with waist-to-hip ratio in men.[25] One study showed the only difference between men with higher vs lower waist-to-hip ratio was a lower sex hormone binding globulin in the former group.[26] In the data presented here, the association of smoking with waist-to-hip ratio was stronger in women than men, consistent with an effect of increased adrenal androgens. Adrenal androgens may have a less obvious effect in men, who have high levels of testicular androgens.

Except for alcohol, there appeared to be little relationship between dietary factors and waist-to-hip ratio other than the inverse relationship with carbohydrates in men. This association could be a chance finding, reflecting the multiple diet comparisons. Alternatively, a high carbohydrate diet may be associated with a healthy lifestyle and exercise.

The apparent sex difference in the relationship of exercise to waist-to-hip ratio is compatible with metabolic studies which show different effects of exercise on fat mass in men compared to women.[27] After a physical training program, obese men show a decrease in total body fat, whereas women do not.[28–30] Since waist-to-hip ratio is highly correlated with percent body fat,[31] exercise could have a greater effect on waist-to-hip ratio in men than in women.

These findings are based on older, White men and women, and may not be generalizable to younger sub-

TABLE 3—Correlations of Waist-to-Hip Ratio with Dietary Variables as Measured by the Harvard-Willett Food Frequency Questionnaire

	Men N = 335	Women N = 402
Total calories		
Unadjusted	.04	.02
Age and BMI Adjusted	.10	.01
% Calories from Alcohol		
Unadjusted	.19§	.15#
Age and BMI Adjusted	.25**	.18†
% Calories from Carbohydrate		
Unadjusted	−.25**	−.06
Age and BMI Adjusted	−.25**	−.03
% Calories from Fat		
Unadjusted	.04	−.08
Age and BMI Adjusted	.01	−.11
% Calories from Protein		
Unadjusted	.08	−.02
Age and BMI Adjusted	.00	−.03
Fiber, dietary		
Unadjusted	−.14*	−.08
Age and BMI Adjusted	−.09	−.00
Caffeine		
Unadjusted	.09	−.05
Age and BMI Adjusted	.07	−.01
Sucrose		
Unadjusted	−.06	−.06
Age and BMI Adjusted	−.05	−.04

*p = .0114, #p = .0023, †p = .0018, §p = .0005, **p = .0001

TABLE 4—Multiple Linear Regression of Behavioral Variables on Waist-to-Hip Ratio

	Men*		Women**	
	β	Standard Error	β	Standard Error
Age (years)	.00066	0.0002	00173	0.0002
BMI (kg/m²)	.00618	0.0005	.00537	0.0005
Alcohol (mean gms/past week)	.00005	0.00001	.00006	0.00002
Cigarettes (mean no.)	.00027	0.0001	.00034	0.0001
Exercise (Y/N)	−.01520	0.0052	−.00288	0.0046

*R^2 = 0.21
**R^2 = 0.16

jects or to minorities. However, the age group studied is that in which "middle-aged spread" is classically manifest. The analyses were based on self-reported behaviors, and it is possible that these were inaccurately reported by the participants. Probable validity of the reported alcohol intake is strengthened by concordance with aspartate aminotransferase activity and HDL-cholesterol in this cohort.[32] Likewise, the validity of self-reported exercise in this cohort is strongly suggested by its concordance with pulse and HDL-cholesterol.[33] In any event, misclassification of self-reported behaviors usually serves to obscure an association, not create one.[34]

The fact that waist-to-hip ratio appears to be determined, at least in part, by behavioral factors has implications for intervention. This risk factor for increased morbidity and mortality may be modifiable by interventions aimed at changing the behaviors which affect it. This would be expected to have a favorable impact on general, and specifically, cardiovascular health.

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The Framingham Disability Study: Relationship of Various Coronary Heart Disease Manifestations to Disability in Older Persons Living in the Community

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Abstract: The relation between coronary heart disease and disability was examined in 2,576 community-dwelling women and men ages 55–88 years. These Framingham Study participants were originally recruited in 1948–51 for an examination of cardiovascular disease. Twenty-seven years later, remaining members of the cohort were interviewed to ascertain physical abilities, and a score on a disability scale was assigned. Multivariate logistic analyses examined disability in relation to uncomplicated angina pectoris (AP), complicated AP, and coronary heart disease other than AP, controlling for possible confounders. In younger and older women and men, uncomplicated and complicated AP were associated with disability. Coronary heart disease other than AP was associated with disability only in the younger men. Congestive heart failure predicted disability only in the women. These results suggest that onset of AP should be recognized as a critical point in the development of disability and that AP is a better predictor of disability than is myocardial infarction or coronary insufficiency. (*Am J Public Health* 1990; 80:1363–1368.)

Introduction

Although the coronary heart disease (CHD) mortality rate in the United States has been declining for two decades,[1] CHD remains the leading cause of death.[2] There are indications that CHD morbidity has not been following the same trend as mortality, and that the number of persons, particularly older persons, disabled from CHD remains high. Estimates of the percentage of persons with ischemic heart disease who were disabled by their condition in 1980 range from 36 percent in 55–64 year old women to 76 percent in men 75 years and older.[3] Heart disease ranks as third among the leading chronic conditions causing limitation of activity; in 1983–85, 5.6 million persons were affected* and this number was higher than a 1980 estimate.**

A better understanding of the CHD-disability relationship may provide leads to primary and secondary preventive health care strategies.[4] Some researchers have sought to identify predictors of disability among persons with CHD.[5–7] The Framingham Study enables us to look at disability in a non-institutionalized population of older persons, to compare physical limitations of those with and without CHD, and to learn more about the relationship of disability to specific CHD manifestations. Our specific question is: among a population of non-institutionalized women and men ages 55–88 years in 1976–78, is disability related to uncomplicated angina pectoris (AP), CHD other than AP, and AP complicated by other CHD? Since congestive heart failure is closely associated with CHD, we will also examine its relation to disability.

Methods

Study Sample

Between 1948 and 1951, the Framingham Study assembled a cohort of 5,209 persons, ages 28–62 years in Framingham, Massachusetts.[8] Information on potential cardiovascular disease risk factors and disease manifestations were obtained every two years by interview, physical examination, and laboratory tests. By December 1979, 31 years after the inception of the study, the 15th examination cycle had been completed.

The Framingham Disability Study (FDS) was designed to yield a quantitative assessment of levels of physical and social disability based on respondents' self-reported information. Surviving members of the Framingham Study reported their current level of functioning in interviews from September 1976 (midway through the 14th examination cycle) through November 1978 (midway through the 15th examination cycle). This time period will be referred to throughout this report as examination 14/15. Descriptions of the prevalence findings on physical disability from this study have been reported previously.[9]

When the Framingham Disability Study interviewing began, 1,649 of the original cohort of 5,209 had died, 218 had moved away and dropped out of the study, 151 had refused to return for examination, and 354 had been missing from the study for no known reason for at least five years.

Of the remaining 2,837 individuals, 183 (6 percent) were not included in the sample of FDS participants: 41 were nursing home residents who were unable to come to the clinic and were therefore excluded; 87 had come into the clinic for their 14th examination before the disability interviewing began and had not come in for their 15th examination by the time the disability interviewing ended; 43 refused to be interviewed; 11 came for their physical examination but were missed by the disability interviewer due to scheduling difficulties and were unable to be rescheduled; and one person died before a rescheduled interview was taken, leaving 2,654 remaining. Members of the original cohort who did not

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*Unpublished table: C3-8501, Prevalence and rate per 1,000 of chronic conditions of six chronic condition lists and ICD codes: United States, 1980. National Health Interview Survey, Center for Health Statistics, November 7, 1986.

**Unpublished table: C-8003, Prevalence of published chronic conditions rates by sex and age, and percent and rates for selected health related characteristics: United States, 1980. National Health Interview Survey, National Center for Health Statistics, undated.

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participate in the FDS for all of the above-mentioned reasons other than death (N = 882), when compared with FDS participants on characteristics at entry to the Framingham Study, were more likely to be female, older, have a higher mean systolic blood pressure, and a higher body mass index (Table 1).

Seventy-eight (3 percent) of the 2,654 participants were excluded from present analyses for the following reasons: 76 persons had missing values in one or more of the items used to calculate a score on the disability scale, and two had missing values in one of the risk factor measures used as covariates in the analyses. There remained 1,511 women and 1,065 men available for this investigation. Ages ranged from 55 to 88 years for the women and from 56 to 86 years for the men.

Definition of Variables

Coronary Heart Disease Manifestations and Congestive Heart Failure: Determination of these end points was based on information from biennial examinations, surveillance of hospital discharge records and information obtained from private physicians. A panel of physician investigators reviewed all pertinent information against minimal uniform criteria before final assignment of a diagnosis.[10] Examination 14/15 disease status was determined using diagnoses made at any time from examination 1 through examination 14/15.

Myocardial Infarction was diagnosed on the basis of unequivocal specific electrocardiographic criteria or elevated serum enzyme levels. This classification included both recognized myocardial infarctions (ones in which onset of symptoms led the individual to seek medical attention) and unrecognized myocardial infarctions (ones in which no symptoms occurred prior to an examination by a physician).

Angina Pectoris: Brief recurrent chest discomfort of up to 15 minutes duration precipitated by exertion or emotion and relieved by rest or nitroglycerin was regarded as angina pectoris (AP) if two physicians interviewing the participant agreed that AP was definitely present. Electrocardiographic abnormalities were not taken into account in making the diagnosis of AP.

Coronary Insufficiency was diagnosed when a history of ischemic chest pain lasting one hour or more was associated with transient ischemic electrocardiographic criteria.

Congestive Heart Failure: A definite diagnosis of congestive heart disease required a minimum of two major criteria (e.g. increased heart size and rales) or one major and two minor criteria (e.g., rales, bilateral ankle edema, and night cough).

TABLE 1—Selected Characteristics of Original Framingham Cohort Members by Participation in the Framingham Disability Study

Characteristics at Entry	Participation Status		95% CI of difference
	No*	Yes	
Number of persons	882	2,654	
Percent female	65.3	58.9	2.72, 10.03
Age (years)	44.0	40.9	2.49, 3.65
Systolic blood pressure (mmHg)	135.2	131.2	2.55, 5.50
Body mass index (kg/m^2)†	25.7	25.4	0.02, 0.64
Cigarettes per day‡	9.2	8.4	−0.02, 1.72

*Persons who died prior to the start of the Framingham Disability Study were excluded from this group.

Number of observations with missing values: † = 4; ‡ = 19.

The following mutually exclusive subgroups of CHD were considered: uncomplicated AP (AP unaccompanied by coronary insufficiency or myocardial infarction); CHD other than AP (coronary insufficiency or myocardial infarction); and complicated AP (AP accompanied by coronary insufficiency, myocardial infarction, or both).

Disability Scale: The measure of disability used was based on three questions drawn from the Rosow and Breslau Functional Health scale:[11] "Are you able to do heavy work around the house, like shoveling snow or washing windows, walls, or floors, without help?" "Are you able to walk up and down stairs to the second floor without help?" and "Are you able to walk half a mile without help? That's about 8 ordinary blocks." The scale ranged from 0 to 3, reflecting the number of activities which the respondent could not do without help. In other words, the higher the score, the more areas of disability were reported; a score of 0 indicated no disability.

Control Variables: To minimize the chance of detecting a spurious relation between the onset of CHD and subsequent disability, we controlled for the effects of co-existing conditions: other cardiovascular diseases, cardiovascular risk factors, and reported health history of other diseases related to disability. Presence of cardiovascular diseases other than CHD, namely stroke and intermittent claudication, were determined in a manner identical to that used to identify CHD and congestive heart failure, by a panel of physicians using uniform criteria after each biennial examination. Detailed descriptions of clinical criteria employed for the diagnoses of these diseases are available elsewhere.[10]

Cardiovascular Disease Risk Factors: We controlled for the long-term presence of hypertension (coded as a categorical variable taking into account anti-hypertensive drug use as well as blood pressure levels), body mass index (weight, kg, divided by height, m^2), cigarette smoking, and blood sugar, the four cardiovascular disease risk factors found in preliminary analysis to be related to the disability scale. For an explanation of how these risk factor measures were calculated, see Pinsky, *et al.*[12]

Other Health Problems: Several open-ended questions solicited information on participants' lifetime health status. Participants were asked first to mention all of the serious health problems they had had over the years, and were told to include "things that were serious at the time, things that remain as potential problems, or things that in any way limit activities at the present time." A two-part question followed: respondents were asked to mention things they used to do that the cannot do now or had to stop or cut down on; and they were asked what health problem caused each limitation. A maximum of 11 self-reported health problems for each individual were noted during the interview and subsequently classified into one of 233 codes. For the present analyses, 131 codes were grouped into 16 categories of health problems. Age-adjusted analyses indicated that six of the 16 categories were related significantly to the disability scale and are included as control variables in these analyses: amputations, arthritis, back problems, gastrointestinal problems, neurological problems (excluding stroke), and respiratory problems. The categories of health problems which were not related to the disability scale were allergies, cancer, ear problems (including hearing impairment), eye problems (including visual impairment), kidney problems, liver problems, hernias, bone injuries and other accidents, and bone problems other than accidents (data available on request to author).

Statistical Analysis

We chose a logistic model for multivariate analysis which is similar to that described by McCullagh, since the disability scale, our dependent variable, is an ordered categorical variable consisting of four levels of increasing severity. In this approach, the probability of being at the ith level or higher is written as a logistic function of the explanatory variables with a indicator term depending on the value of i. A modification of McCullagh's analysis permits inclusion of continuous explanatory variables. Maximum likelihood was used for estimation, and odds ratios were calculated as the exponent of the appropriate logistic regression coefficient. This model assumes that the underlying odds ratios for the three ordered comparisons of scores on the dependent variable (1 versus 0, 2 versus 1, and 3 versus 2) are equal. We used the logistic procedure in the Statistical Analysis System available from the SAS Institute.[14]

In a previous report, a different disability scale and disease were analyzed separately for women and men, controlling for age.[15] In this report, separate analyses were conducted for two age categories among women and men in an effort to provide additional insights. Furthermore, the lack of a linear relationship between disability and age[9] suggests the relevance of conducting age-specific analyses, rather than putting age in the multivariate model. Each regression included the following control variables: cardiovascular risk factors (i.e. hypertension, cigarette smoking, body mass index, and blood sugar); intermittent claudication and stroke; and six other health problems, (i.e. amputations, arthritis, back problems, gastrointestinal problems, neurological problems, and respiratory problems).

Results

Mean age was 68.3 years for 1,511 women and 67.2 years for 1,065 men. The percentage distribution of scores, from 0 to 3, on the disability scale were, respectively, 61.0, 26.6, 8.2, and 4.2 for women and 78.1, 16.6, 3.6, and 1.7 for men. Not only did a greater percentage of women report disabilities compared to men, but they reported being disabled in more activities than men.

Table 2 displays the age- and gender-specific prevalence of scores greater than 0 on the disability scale by CHD and congestive heart failure status. In all gender and age strata, disability was more prevalent in persons with CHD or congestive heart failure than in persons free of CHD or congestive heart failure. Persons with AP were more likely to be disabled than those with CHD manifestations other than AP. Congestive heart failure was more disabling in women than men.

Gender- and age-specific multivariate logistic analyses examined the relationship between the disability scale and the three CHD manifestations and congestive heart failure. Using the results of these analyses, odds ratios were calculated, representing a comparison of the disability status of persons with the manifestation to persons free of all disease categories included in the model (Table 3). An odds ratio of one means that those with the manifestation have the same chance of having a higher score on the disability scale as persons free of all disease categories included in the model, within each particular gender and age subgroup. For both women and men, AP both uncomplicated and complicated is strongly related to the disability scale in both age groups, although the relationship weakens with age. CHD other than AP is related to the disability scale only in the younger men. Congestive heart failure is related to disability only in the younger women.

Table 4 presents the results of analyses similar to those in Table 3, except the model also includes terms for other cardiovascular diseases, cardiovascular risk factors, and the self-reports of non-cardiovascular health conditions. The inclusion of these additional covariates did little to affect the results of the analyses. When comparing Table 4 to Table 3, one notes only minor shifts, most of which are downward, in the magnitude of the relationships between disability and disease.

Myocardial infarction is often the event of interest in epidemiological studies and is a much more frequent diagnosis when compared to coronary insufficiency. If we exclude from our pool of persons under study the 56 with coronary insufficiency, our CHD categories become uncomplicated AP, uncomplicated myocardial infarction, and AP complicated by myocardial infarction. Analyses based on these definitions resulted in findings similar to those which included coronary insufficiency. When the three levels of disability were combined, making the dependent variable dichotomous, the pattern of significant relationships did not change at all. For most associations, the strength of the relationship was greater when compared to the analyses using the multichotomous measure.

Discussion

Although our results indicate that AP is a better predictor of disability than other CHD, we were unable to control

TABLE 2—Percent of Women and men with Disability* at Examination 14/15 by Coronary Heart Disease and Congestive Heart Failure Status and Age

	Women			Men			Women			Men		
	N	% with Disability	95% CI†	N	% with Disability	95% CI	N	% with Disability	95% CI	N	% with Disability	95% CI
	Ages 55–69 years			Ages 56–69 years			Ages 70–88 years			Ages 70–86 years		
No Coronary Heart Disease or Congestive Heart Failure	829	24.7	21.8, 27.7	574	9.4	7.0, 11.8	471	48.8	44.3, 53.3	275	26.9	21.7, 32.2
Uncomplicated Angina Pectoris	44	65.9	51.9, 79.9	39	46.2	30.5, 61.8	64	81.3	71.7, 90.8	38	50.0	34.1, 65.9
Coronary Heart Disease other than Angina Pectoris	14	50.0	23.8, 76.2	40	32.5	18.0, 47.0	26	61.5	42.8, 80.2	33	39.4	22.7, 56.1
Complicated Angina Pectoris	23	69.6	50.8, 88.4	42	66.7	52.4, 80.9	19	94.7	84.2, 100	21	66.7	46.5, 86.8
Congestive Heart Failure	15	80.0	60.0, 93.3	7	42.9	14.2, 71.4	25	88.0	80.0, 100	14	57.1	31.2, 83.1

*Defined as a score of 1, 2, or 3 on the disability scale.

†CIs were calculated as follows: when both n × p and n × q were greater than 5, the normal approximation was used; otherwise the exact binomial was used.

TABLE 3—Association between the Disability Scale and Coronary Heart Disease Manifestations and Congestive Heart Failure by Sex and Age, from Logistic Regression Analyses (Terms in the model: the four disease categories listed below and age)

Disease Category	Women		Men		Women		Men	
	Odds Ratio	95% CI	Odds Ratio	95% CI	Odds Ratio	95% CI	Odds Ratio	95% CI
	Ages 55–69 years (N = 918)		Ages 56–69 years (N = 695)		Ages 70–88 years (N = 593)		Ages 70–86 years (N = 370)	
Uncomplicated Angina Pectoris	5.87	3.30, 10.43	6.71	3.43, 13.11	2.27	1.39, 3.70	2.56	1.31, 5.01
Coronary Heart Disease other than Angina Pectoris	1.70	0.60, 4.80	4.44	2.16, 9.10	1.53	0.72, 3.23	1.83	0.85, 3.95
Complicated Angina Pectoris	5.10	2.30, 11.31	14.00	7.46, 26.29	7.53	3.33, 17.02	4.91	2.10, 11.47
Congestive Heart Failure	9.09	3.34, 24.76	0.97	0.20, 4.58	2.07	0.97, 4.40	2.96	0.98, 8.94

TABLE 4—Association between the Disability Scale and Coronary Heart Disease Manifestations and Congestive Heart Failure by Sex and Age, from Logistic Regression Analyses (Terms in the model: the four disease categories listed below, age, and additional covariates*)

Disease Category	Women		Men		Women		Men	
	Odds Ratio	95% CI	Odds Ratio	95% CI	Odds Ratio	95% CI	Odds Ratio	95% CI
	Ages 55–69 years (N = 918)		Ages 56–69 years (N = 695)		Ages 70–88 years (N = 593)		Ages 70–86 years (N = 370)	
Uncomplicated Angina Pectoris	4.86	2.66, 8.85	5.39	2.6, 11.03	1.98	1.20, 3.27	3.28	1.62, 6.66
Coronary Heart Disease other than Angina pectoris	1.45	0.51, 4.14	3.55	1.63, 7.76	1.19	0.55, 2.58	1.76	0.78, 3.98
Complicated Angina Pectoris	5.27	2.30, 12.10	11.02	5.67, 21.42	5.92	2.48, 14.11	5.65	2.29, 13.99
Congestive Heart Failure	6.52	2.32, 18.31	0.99	0.19, 5.11	1.80	0.84, 3.86	1.79	0.56, 5.71

*Additional covariates: hypertension, cigarette smoking, body mass index, blood sugar, stroke, intermittent claudication, amputations, arthritis, back problems, gastrointestinal problems, neorological problems, and repsiratory problems.

for selection factors which could play a role in this study, factors somewhat parallel to the healthy worker effect. Participants are very possibly as a whole healthier in general than nonparticipants. As the longitudinal study progresses, widowhood, geographical relocation, and entrance into institutionalized care facilities probably account for differences in the health status of participants and nonparticipants. For example, one can surmise that the older male participants with congestive heart failure have less severe cases than the surviving cohort members who did not appear for examination 14/15; the milder nature of their disease may partially account for the lack of a relationship with disability.

The loss of severe cases through mortality is probably more relevant among the older persons. The loss of participants results in a smaller population and therefore a possible power problem in our analyses of the 70–88 year olds. It is also important to consider that as age increases, disability due to other conditions for which we have not controlled for such as depression or senility, may interfere increasingly with the assessment of CHD-related disability. Another limitation of our analyses is the absence of consideration of psychosocial variables which may influence physical functioning and consequently could confound our results.

In spite of factors which could attenuate the associations between CHD and disability, suffering from AP was a predictor of disability in each gender and age subgroup. This finding provides epidemiological support for what is commonly observed in clinical practice: once a person develops AP, it chronically affects physical function, regardless of the presence of CHD risk factors, stroke, intermittent claudication, and self-reported health conditions other than cardiovascular disease. Complicated AP was a stronger predictor of disability than uncomplicated AP. This may be explained in part by the differing severity of AP in those with and without other CHD and in part because of limited myocardial reserve imposed by myocardial infarction. That these relationships were generally stronger in men than women could reflect the fact that men suffer more severe disease than women, or that men may initially be more active, enabling their disease to have a greater potential for being more limiting.

The Framingham Study protocol dictated that once an individual was diagnosed as having AP, the individual was never re-evaluated for presence of AP. This limitation prohibited us from identifying those persons who were suffering from AP at examination 14/15; our classification of AP was essentially AP at any time from examination 1 through examination 14/15. We suspect that had we been able to use a determination of current AP sufferers, the relationship to disability would have been even stronger than our analyses indicated.

In sharp contrast to the strongly positive relationship between AP and disability, CHD other than AP was a significant predictor of disability only in the younger men. The finding is consistent with the explanations mentioned above for comparisons between older and younger participants, and between women and men. In these 40 women and 73 men diagnosed as having CHD other than AP, the mean amount of time that elapsed from the most recent event to the time of the disability measurement was greater for the older groups. This fact further helps to explain why the relation between disease and disability are weaker in the older groups; our data seem to indicate that initial disability with acute manifestations probably lessens or disappears with time. This interpretation is consistent with the results of a study of 9,385 men and women aged 18–103 years.[16] In this study a physical functioning measure, based on self-reported capacity to perform a variety of physical activities, was significantly related to a multivariate model to occurrence of myocardial infarction within the prior 12 months. Angina was also found to be significantly related to physical functioning.

The ostensibly puzzling difference between men and women in their congestive heart failure-disability relationships (significant in women but not in men) may be understood better when the relationships between CHD and congestive heart failure are examined. Among the men, 85.7 percent of those with congestive heart failure also had CHD, whereas only 47.5 percent of the women with congestive heart failure were diagnosed as also having CHD. One could hypothesize that in the 21 men with congestive heart failure, their CHD was so disabling that additional disability could not be detected. Furthermore, the very low congestive heart failure prevalence in the men probably works against the detection of a relation with disability.

While we found only one other study comparing disability in persons with and without CHD,[16] other studies have examined predictors of cardiac disability in patient-based populations, the results of which are largely complementary to ours. In a study of men ages 35–77 years with CHD, an activity score was calculated from a self-administered instrument and included such items as sports, walking, sex, and heavy housework; regression analysis on 98 of these men showed that angina was related to total activity score, but past myocardial infarction history was not so related.[6] Chirikos and Nickel studied 976 men and women (age range not identified) who were hospitalized for acute coronary attacks and found in their multivariate analyses that presence of cardiac and chronic diseases, including angina, was significantly related to restoration of functional capacity as determined by telephone interview at 6, 18 and 24 months post-hospitalization.[7] They also noted that gender was not significantly related to the restoration of functional capacity. Results from Hlatky, *et al*, are very different; in their study, a self-administered questionnaire was used to determine whether the study participants were not working because of disability.[5] Among 814 men younger than 60 years with coronary artery disease, an anginal pain scale was not related to work disability, but history of myocardial infarction was so related. This study utilized a very different type of disability measure, which might partially explain why its results differ from ours and above-mentioned studies.

In addition to the selectivity factor previously noted, another limitation of the study design warrants mention. Our study question assumes that disability developed subsequent to disease. Because assessment of physical disability status was made at a single point in time (examination 14/15), it is possible that some persons were disabled at the start of their participation in the Framingham Study or otherwise preceded the diagnosis of CHD. The items comprising the disability scale have been asked at a later Framingham examination; analyses which include these data will hopefully enable a study of change of disease status with change in disability status.

In conclusion, our analyses revealed a striking difference in the disabling effects of AP and other CHD. AP, whether complicated or not, was predictive of disability, whereas other CHD was not, except in the youngest women. This finding is in contrast to the relative roles of CHD manifestations when the outcomes of interest are further morbidity and mortality; that is, AP is less predictive of CHD morbidity and mortality than other CHD.[17] The interactions between disability and disease are complex, and it is important to keep in mind that the presence of disease may not necessarily contribute to disability, further morbidity, and mortality to a similar extent. Finally, age is another important factor to take into consideration, as our analyses indicated differences in the strength of the relationship of disability to CHD in different age groups.

These results further the cause of preventive cardiology. Our findings provide population-based evidence to back up clinical knowledge of consequences of CHD. Motivation for control of cardiovascular risk factors is not only the avoidance of well-documented morbidity and mortality from CHD other than angina pectoris; our study demonstrates clearly and provides quantification of the disabling nature of angina pectoris.

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The Effects of Psychosocial Work Organization on Patterns of Cigarette Smoking among Male Chemical Plant Employees

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Abstract: We tested the hypothesis that job strain (the combination of high psychological job demands and low work control) is positively associated with smoking prevalence and intensity in a study group of 389 males employed in a chemical plant, using a self-administered questionnaire. In a logistic regression analysis which controlled for a number of sociodemographic factors, job strain was not found to be associated with smoking cessation. However, among smokers, those in higher-strain jobs smoked more heavily than those in lower-strain positions (OR 1.70, 95% CI = 1.10, 2.61) and were more likely to have increased the amount they smoke (OR 3.72, 95% CI = 1.92, 7.17). (*Am J Public Health* 1990; 80:1368–1371.)

Introduction

Cigarette smoking is a harmful habit, not only in terms of its health consequences but also with respect to its financial impact. Workers who smoke are said to have higher rates of absenteeism, hospitalization, illness, and job-related accidents; each employee who smokes is estimated to cost business $300 to $1000 a year.[1] Corporate concern about such facts is indicated by the current proliferation of worksite anti-smoking policies and cessation programs. However, in contrast to the extensive documentation of smoking's effects on employment, the converse of this relationship—how work may influence smoking behavior—remains largely ignored.

Overall, blue-collar workers are much more likely to smoke and less likely to quit smoking.[2] The differential is somewhat smaller for women than for men. While the proportion of persons who have made a serious attempt to quit does not differ substantially between white- and blue-collar workers (64 vs 59 percent, respectively), white-collar workers are more likely to have succeeded (48 vs 35 percent success).[3]

The relation between occupation and smoking may be mediated by the psychosocial work environment. Despite a lack of consistency in definitions and measurement, research has generally found work-related stress to inhibit cessation[4,5] and increase intensity of smoking.[4-7] Workplace social support may affect smoking behavior negatively or positively, depending on the prevalence of smoking and attitudes towards quitting among co-workers.[4,8] In addition, studies of work socialization have indicated that low job control contributes to psychological characteristics such as poor self-esteem and external locus of control,[9,10] which have been associated with a lower likelihood of quitting smoking.[11,12]

Karasek and his colleagues have defined job strain as the combination of high psychological demands with low decision-latitude or control.[13] Research in Sweden and the United States using this model has found high-strain work—where job demands are high and there is little opportunity for control—to be associated with an increased risk of cardiovascular disease (CVD), independent of smoking.[13-18] More recently, a modified version of this formulation which includes social support has identified a high CVD risk group of strained and socially isolated workers.[19,20] If job stress or strain also increases smoking, it would pose a double risk to cardiovascular health: directly, through physiological mechanisms, and indirectly, through behavioral risk factors such as smoking.

The central hypothesis of the present study was that job strain, defined as a high ratio of psychological demands to control, is negatively associated with smoking cessation and positively associated with both smoking intensity and an increase in smoking over time. A secondary objective was to explore the relationship between workplace social support and smoking behavior.

Methods

Study Group

The population surveyed consisted of the approximately 800 employees at a chemical manufacturing plant in the northeastern United States. Two years prior to the study, the plant had established an on-site "Wellness Center," consisting of exercise facilities staffed by two health educators.

Each employee was mailed a copy of the questionnaire described below; after two mailed follow-ups to non-respondents, a final response rate of 60 percent among full-time employees was obtained. From the 466 completed questionnaires, 23 females and 54 males under the age of 40 were excluded, since their work histories were substantially different from those of the majority of the workforce and their numbers too small to allow for meaningful subanalysis. Thus, all analyses were conducted on a study group of 389 male respondents ages 40 and above. The typical study group member was White (93 percent), married (85 percent), and had a high school (51 percent) or more than high school (29 percent) education. Over half of respondents reported earning between $30,000 and $40,000 annually.

Non-respondents were less likely than respondents to have registered with the exercise center (28 percent vs 39 percent), and less likely to be salaried (22 percent vs 34 percent) (as opposed to being paid hourly).

Table 1 presents the distribution of job titles in the study group, classified in seven occupational categories. Almost three-fourths were paid on an hourly basis. Half of the group, mostly hourly employees, worked shifts. The average length of time with the company was just over 25 years.

Questionnaire

The questionnaire collected information on job characteristics, health behavior, and demographics.

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TABLE 1—Distribution of Occupations in Male Study Participants

Occupational Category	N	%
Unskilled workers	26	(6.8)
Semi-skilled workers, machine operators	151	(39.2)
Skilled manual workers	87	(22.6)
Clerks, technicians	74	(19.2)
Administrative personnel, minor professionals	17	(4.4)
Business managers, lesser professionals	18	(4.7)
Higher executives, major professionals	12	(3.1)
No answer	4	
Total	389	

Job Characteristics: This section was based on Karasek's Job Content Instrument (JCI),* with minor modifications. The JCI was designed to measure the content of work tasks in a way which is applicable to all jobs in the United States. Its main focus is the psychological and social structure of work. The six job characteristic scales formed from this questionnaire assess 1) job control, 2) psychological demands, 3) physical demands, 4) job insecurity, and 5–6) social support from co-workers and from supervisors. In addition, a seventh variable measuring job strain was created by dividing the standardized score on the psychological demands scale by the standardized control score. In accord with the job strain hypothesis, the higher the demand/control ratio, the higher the level of strain. (Details of scale content and construction are in the Appendix.)

Smoking Behavior: From the questions on smoking behavior, three dichotomous smoking variables were created.

1) Smoking cessation (smokers vs ex-smokers);
2) Smoking intensity (less vs more than a pack a day);
3) Change in smoking habits since starting to work for the company (started smoking or increased amount vs no change or decreased).

Statistical Analysis

For the bivariate analyses, simple odds ratios (ORs) measured the strength of the associations, with corresponding 95 percent confidence intervals (95% CIs) calculated using Woolf's method.[21]

Multivariate analysis of the relations between job characteristics, demographic factors, and smoking behavior was conducted. In a logistic regression analysis, a stepwise backward selection procedure was used to determine which of the job characteristics and/or demographic variables were predictors of each smoking behavior variable with the inclusion level set at 0.10.

Standardized ORs (SORs) with 95 percent confidence intervals were computed from the standardized logistic coefficients, following the method proposed by Rosenmann, *et al.*[22] To indicate a negative association, a [−] sign is used, rather than the reciprocal of the SOR. The following variables were initially entered into the logistic model: job strain (demand/control ratio), job insecurity, physical demands, co-worker support, supervisor support, shiftwork (1 = work shifts; 0 = do not), marital status (1 = divorced, 0 = other), education, income, age, and race (1 = White, 0 = other).

To further examine the relation between job characteristics and behavior, multiple contingency tables were formed by stratifying the data into categories of each of the other variables retained in the logistic model, alone and in combination. From these tables, an estimate of a uniform OR over all strata was calculated using Mantel-Haenszel procedures.[23] The heterogeneity of the ORs across strata was determined by a likelihood ratio test of heterogeneity.[24] Confidence intervals around the Mantel-Haenszel point estimates were then calculated using Miettinen's test-based interval estimation procedure.[25] This analysis produced generally similar results to logistic regression, with all ORs heterogeneous across strata; thus, multiple contingency tables are discussed here only where the stratum-specific ORs are of interest.

Results

Almost half (49 percent) of the 389 study participants had quit smoking and 31 percent were current smokers. Half of the former smokers (50 percent) had quit 11 or more years earlier; just over one-third (36 percent) had quit between two and 10 years earlier and 14 percent had quit within the preceding year.

Almost half of the 117 smokers (46 percent) reported smoking more than 20 cigarettes per day; a slightly smaller proportion (41 percent) smoked 10 to 20 a day, and 13 percent smoked fewer than 10. In response to the question of whether their smoking habits had changed since starting to work for this company, 52 percent reported no change, 35 percent an increase, 10 percent a decrease, and 3 percent said they had only begun to smoke since starting to work.

Bivariate Analyses

Bivariate analyses found that, as predicted, smokers in high-strain jobs are much more likely to smoke heavily and to report an increase in smoking since starting to work for the company; those in low-strain jobs are also more likely to have quit smoking, although this association is weaker (Table 2).

There is a steady increase in the proportion of heavy smokers as job strain increases. Similarly, the proportion of smokers reporting an increase in smoking grows progressively with increasing job strain. Furthermore, the proportion of smokers who have decreased or maintained their smoking is inversely related to job strain, with a significant number reporting a decrease in smoking only at the lowest level of strain.

Multivariate Analyses

Controlling for demographic and other job characteristic variables through logistic regression reduces the association between strain and cessation (Table 3), but the relations between job strain and intensity of smoking and between job strain and increase in smoking remain strong (Tables 4 and 5). In each case, adjusting for the demographic variables remain-

TABLE 2—Bivariate Relations between Smoking Behavior Variables and Job Strain (Low vs High)

Smoking Behavior Variables	Odds Ratio (95% CI)
Cessation (former vs current smokers)	1.71 (0.92, 3.17)
Intensity (≤20 cigarettes per day vs >20)	3.86 (1.35, 10.99)
Change in smoking habits (increased/started vs no change/decreased)	7.78 (2.28, 26.50)

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*Job/Heart Project: Job Content Questionnaire and User's Guide, Revision 1.1. New York: Columbia University, March 1985. Unpublished manuscript.

TABLE 3—Smoking Cessation: Variables Retained in Logistic Model

Variables	Standardized Odds Ratio (95% CI)
Age	−1.45 (−1.14, −1.86)
Divorce	1.37 (1.07,1.75)
Co-worker Support	1.35 (1.05,1.77)
Job Strain	1.17 (0.90,1.51)

TABLE 4—Smoking Intensity: Variables Retained in Logistic Model

Variables	Standardized Odds Ratio (95% CI)
Job Strain	1.70 (1.10,2.61)
Education	−1.61 (−1.05, −2.47)
Income	1.50 (0.97,2.37)

TABLE 5—Change in Smoking Habits: Variables Retained in Logistic Model

Variables	Standardized Odds Ratio (95% CI)
Job Strain	3.72 (1.92,7.17)
Age	−2.16 (−1.32, −3.53)
Education	−2.09 (−1.22, −3.55)
Income	1.70 (1.00,2.89)

ing in the model strengthens the association between job strain and smoking behavior.

The interrelations between smoking cessation, co-worker support, and demographic variables were examined further in multiple contingency table analysis. Overall, co-worker support is negatively associated with having quit smoking. Adjusting the relation between co-worker support and quitting for both education and age reveals some interesting interactions. Co-worker support has little impact on quitting among younger men, regardless of education. Among older men with a high school education or less, co-worker support is associated with smoking (OR 3.13, 95% CI = 1.33, 6.91), while for older men with more education, it is unstably associated with quitting (OR 4.40, 95% CI = 0.82, 20.37).

Discussion

Our failure to find a relation between job strain and quitting may have been due to the cross-sectional nature of the study: half of the ex-smokers had quit more than 10 years earlier and one-fourth 20 or more years earlier, at which time their job characteristics might have been quite different. Another factor which could affect this association is awareness of occupational exposure to carcinogens. In the 1960s and '70s, a number of workers at this plant had developed lung cancer as a result of their in-plant exposure. Most of the older production workers were at the plant during that time and may have been motivated to quit smoking by the deaths of their co-workers.

As hypothesized, smokers who experience an excess of psychological demands relative to control tend to smoke more heavily and are more likely to report an increase in smoking over time than those in jobs where demands and control are better balanced. The question on which the association between job strain and increase in smoking is based relied on self-reported assessment of change over a long period of time. Smokers had worked for the company from nine to 42 years, half between 21 and 29 years. However, the accuracy of the report is suggested by the fact that those who reported an increase in smoking were much more likely to smoke over a pack a day (OR 2.81, 95% CI = 1.24, 5.97).

The most important occupational predictor of smoking cessation in this group was co-worker support. While the overall relation is not strong, the stratified analysis by age and education indicates statistical interactions. For older smokers with a high school education or less, strong co-worker support was associated with a lower likelihood of having quit smoking. For those with more education, the opposite is true: those with strong social support were *more* likely to have quit.

The finding that the effect of social support depends on educational level is consistent with the fact that in this study group, smoking was more prevalent among less-educated older men than among those with more education (31 vs 9 percent current smokers, respectively) and that the former were more likely to report that most of the people with whom they spend time do not care whether they smoke, while the latter tended to believe that most people would like them to quit. Other studies also suggest that higher education is associated with more negative attitudes toward smoking.[26] However, a plausible alternative explanation for this finding is that smoking status affects co-worker support: those who work with a group of smokers may fit in better if they also smoke, while being one of a minority of smokers could impair relations with non-smoking co-workers.

If job strain does increase the intensity of smoking, attempts to encourage smokers in high-strain jobs to quit smoking may be hampered by the nature of their work. Thus, modifying job structure so as to increase control and thereby decrease strain could enhance the success of cessation efforts in this population. This recommendation may seem unnecessary and inappropriate if smoking is simply considered a personal behavior controlled by the individual. However, as Syme has pointed out, the systematic patterning of smoking rates in society strongly suggests that social and cultural forces play an important role in this behavior.[26]

APPENDIX

Construction of Job Characteristic Scales

Control

This scale sums responses to 15 items: six items measure skill discretion (degree to which the job involves learning new things, repetitiveness, creativity, varied tasks, and development of the individual's special abilities), seven items measure decision authority (individual's ability to make decisions about his/her own job, to influence the work group, and to influence company policy), and two items measure closeness of supervision (alpha = .85).

Psychological Demands

This scale sums responses to 10 items assessing how fast and how hard the respondent has to work, hectic nature of the job, whether there is enough time to get the job done, frequency of work under time pressure, amount of work and level of concentration required, presence of conflicting demands, and how often tasks are interrupted or work is slowed down by waiting for other people or departments (alpha = .78).

Job Insecurity

This scale sums responses to six items: level of job security, prospects for career development and promotion, future value of skills, possibility of lay-off in recent past and near future, and ability to keep up with work (alpha = .69).

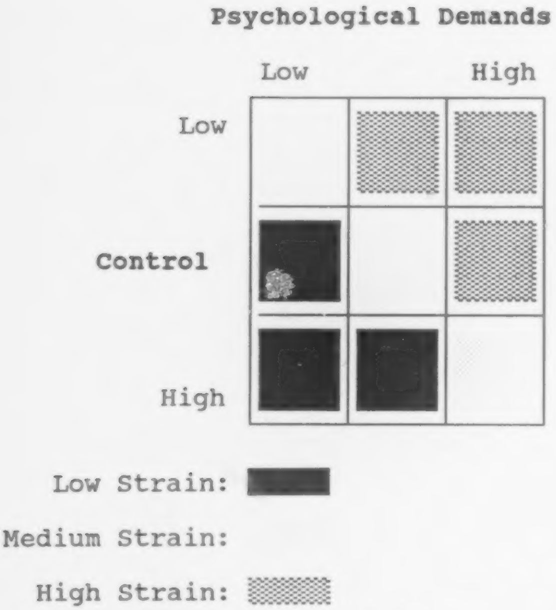

FIGURE 1A—Construction of Three-Level Strain Variable

Physical Demands

This scale sums responses to five items measuring physical exertion (degree to which job involves fast and continuous physical activity, intensive physical effort, and lifting) and ergonomic strain (how often body or head and arms must be held in awkward position) (alpha = .91).

Co-worker Social Support

This scale sums responses to four items measuring co-workers' competence, helpfulness in getting the job done, friendliness, and degree to which they take a personal interest in the respondent (alpha = .71).

Supervisor Social Support

This scale sums five items measuring the supervisor's concern for subordinates' welfare, competence, helpfulness in getting the job done, hostility, and degree to which he/she pays attention to what respondent says (alpha = .84).

Each of these scales was standardized by subtracting the mean from the raw score and dividing the result by the standard deviation; a constant was then added to avoid negative scores. To form contingency tables for bivariate and multivariate analyses, these standardized scores were divided into approximate tertiles (except for the two social support variables which could only be divided roughly at the median, because of their distribution).

Job Strain

In addition to the continuous job strain measure (demand/control ratio), a three-level ordinal form was created from a nine-cell matrix constructed from the three levels of control and psychological demands. Each of these nine cells was then classified into one of three levels of strain, based on the Demand-Control Model, which hypothesizes a dimension of strain running diagonally from the lowest level of demands and highest level of control to the opposite extreme (Figure 1A).

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Use of Fat-Modified Food Products to Change Dietary Fat Intake of Young People

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Abstract: Food purchasing and preparation practices were modified in two boarding high schools to increase the polyunsaturated-to-saturated fat ratio (P/S) of the diet of students by changing food products rather than attempting to change eating behaviors. During years when fat-modified products were served, the P/S of males increased by 75 percent, versus a decrease of 6 percent during control years. For females, P/S increased by 53 percent during intervention years, versus an increase of 6 percent during control years. (*Am J Public Health* 1990; 80:1374–1376.)

Introduction

In an effort primarily to decrease the risk of premature cardiovascular disease, many health promotion organizations currently advise that all Americans, including young people, modify the amount and type of fat in their diets.[1-3] However, the traditional, one-on-one counseling approach to changing eating behaviors is difficult and time-consuming and is an inefficient method for changing the dietary practices of an entire population. An alternate approach is to change the fat content of food products. This would appear to be particularly relevant today, since such a large proportion of foods consumed in the United States are manufactured, processed, or prepared by the industry, rather than prepared from scratch in the home. This limits the control that individuals have over the amount and type of fat that is contained in the foods they consume.

Studies were carried out at two boarding high schools in New England to determine the impact of modifying fats in food products served in the schools' dining halls on the polyunsaturated-to-saturated fat ratio (P/S) of the diets of students. The goal was to determine the effects on the total intake of nutrients of a "passive" intervention, i.e. an intervention at the level of food production and preparation only. Foods with a higher P/S ratio were purchased and changes were made in food preparation practices in the schools' kitchens, but students were not advised to modify their eating behaviors. This paper reports the effects on students' overall dietary fat intake when the fat-modified products were introduced into the dining halls of each of the two schools over a school year.

Methods

The project was carried out at Phillips Exeter Academy in Exeter, New Hampshire and Phillips Academy in Andover, Massachusetts during 1986–88. School food service workers at each institution were trained to procure and prepare food products in which some of the saturated fat content was replaced by polyunsaturated fat.[4,5] It was sought to avoid alteration of the total fat content of food products. The general intent was to make only those modifications that were feasible in terms of product availability, acceptability, and cost, at the time of the study, for an institutional food service operation.

The study design was that of a longitudinal investigation with food product modification being carried out in each of two schools in alternate school years. After the first month of each school year, modifications of food purchasing and preparation practices were carried out in the school which was serving as the intervention school for the year, while the other school continued to serve usual food products. Some modification was carried out for almost two-thirds of fat-containing foods served. While students were aware that some foods were being prepared using different types of fat, they were not told and, in general, could not tell which items had been modified.

For each of the two years, all students at both schools who were taking selected courses in biology or chemistry (approximately 200 at each school each year) were trained and asked to furnish food diaries at specified times during the year to monitor the effects of the food service changes on their intakes of calories and specific nutrients. All foods ingested were recorded, including those foods that were obtained outside of the dining halls (which furnished 28 percent of calories).[6] Comparisons were made between trends in fat intake in the two schools between a baseline period in the fall (when both schools were serving usual products) and a follow-up period in the spring, when the intervention school students were being served fat-modified products. Analysis of food diaries was performed by trained registered dietitians using the "Food Finder" program at the Center for Clinical Computing, Beth Israel Hospital, Boston, MA.[7]

A total of 6,436 food diaries were submitted during the control and intervention years at the two academies. Data provided by all students completing food diaries during a period were used to calculate mean values for that period.

Results

Baseline and follow-up dietary data for males and females are given in Tables 1 and 2, respectively. At baseline, all groups were consuming approximately 35 percent of their calories from fat. During intervention years, the P/S ratio of the students' diet increased by 75 percent (from 0.53 to 0.93) for males and by 53 percent (from 0.64 to 0.98) for females. During control years, the P/S ratio decreased by 6 percent for males and increased by 6 percent for females.

Discussion

The present study was designed to test the feasibility of changing the P/S ratio of the diet of boarding school students

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TABLE 1—Baseline and Follow-up Dietary Data among Males, by Intervention Status (Mean ± SD)

	Control			Intervention			Intervention Effect (Intervention-Control)	
	Baseline (n = 197)	Follow-up (n = 109)	Diff. (%)	Baseline (n = 198)	Follow-up (n = 98)	Diff. (%)	Effect (%)	(95% CI)
Calories	2831 ± 801	2603 ± 786	−8	2835 ± 802	2420 ± 688	−15	−7	(−14, +1)
Total Fat (%kcal)	35.5 ± 5.0	33.6 ± 5.6	−5	34.5 ± 5.4	31.9 ± 6.5	−8	−2	(−7, +3)
Total Fat (gm)	112.1 ± 37.7	98.7 ± 34.5	−12	110.9 ± 41.3	86.6 ± 30.3	−22	−10	(−19, −1)
Polyunsaturated fat (%kcal)	6.1 ± 1.8	5.5 ± 1.7	−10	5.7 ± 1.8	7.9 ± 3.4	+39	+48	(+34, +63)
Polyunsaturated fat (gm)	19.3 ± 7.6	16.2 ± 6.9	−16	17.8 ± 7.4	21.1 ± 9.2	+19	+35	(+21, +48)
Monounsaturated fat (%kcal)	12.5 ± 2.4	12.1 ± 2.4	−3	12.5 ± 2.4	10.9 ± 2.6	−13	−10	(−15, −4)
Monounsaturated fat (gm)	39.8 ± 14.4	35.6 ± 13.1	−11	40.5 ± 16.9	29.7 ± 11.5	−27	−16	(−25, −8)
Saturated fat (%kcal)	12.3 ± 2.6	12.0 ± 2.6	−2	12.2 ± 2.6	9.6 ± 2.8	−21	−19	(−25, −13)
Saturated fat (gm)	39.5 ± 14.9	35.1 ± 13.1	−11	37.8 ± 15.6	26.0 ± 11.5	−31	−20	(−29, −11)
P/S Ratio	0.54 ± 0.20	0.51 ± 0.22	−6	0.53 ± 0.21	0.93 ± 0.45	+75	+81	(+60, +102)

TABLE 2—Baseline and Follow-up Dietary Data among Females, by Intervention Status (Mean ± SD)

	Control			Intervention			Intervention Effect (Intervention-Control)	
	Baseline (n = 188)	Follow-up (n = 130)	Diff. (%)	Baseline (n = 191)	Follow-up (n = 130)	Diff. (%)	Effect (%)	(95% CI)
Calories	2040 ± 552	1898 ± 649	−7	2063 ± 519	1773 ± 581	−14	−7	(−14, −0)
Total Fat (% kcal)	34.9 ± 5.5	34.1 ± 6.0	−2	34.2 ± 5.9	30.7 ± 6.3	−10	−8	(−12, −3)
Total Fat (gm)	80.9 ± 27.2	73.7 ± 31.6	−9	81.1 ± 27.2	62.1 ± 26.9	−23	−15	(−23, −6)
Polyunsaturated fat (%kcal)	6.5 ± 2.3	6.5 ± 2.6	0	6.4 ± 2.0	7.8 ± 2.9	+22	+22	(+11, +33)
Polyunsaturated fat (gm)	14.5 ± 5.8	13.2 ± 5.1	−9	14.5 ± 5.9	15.7 ± 7.6	+8	+17	(+6, +29)
Monounsaturated fat (%kcal)	11.7 ± 2.5	11.3 ± 2.6	−3	11.6 ± 2.6	9.8 ± 2.5	−16	−12	(−18, −6)
Monounsaturated fat (gm)	27.2 ± 10.3	24.6 ± 11.8	−10	27.5 ± 10.3	20.1 ± 10.3	−27	−17	(−27, −8)
Saturated fat (% kcal)	12.1 ± 2.9	11.7 ± 3.3	−3	11.8 ± 2.8	9.2 ± 2.7	−22	−19	(−25, −12)
Saturated fat (gm)	28.0 ± 10.7	25.9 ± 13.4	−8	26.6 ± 10.2	18.6 ± 9.7	−30	−23	(−33, −13)
P/S Ratio	0.62 ± 0.28	0.66 ± 0.40	+6	0.64 ± 0.25	0.98 ± 0.42	+53	+47	(+31, +62)

by modifying the foods served rather than by changing student eating behaviors. The modified products were very well-accepted by students. While the estimated effect of the intervention on the diets of students is based on their self-reports of intake, the students had not been told to change their dietary habits or fat intake and thus were unlikely to be trying to please the investigators by reporting what was believed to be desired. Also, in general, they did not know which products had been modified. The study demonstrated that the modification of approximately two-thirds of the fat-containing food products served in the schools' dining halls reduced total saturated fat intake by over 20 percent and resulted in a P/S ratio approaching 1.0, the level currently recommended by most health agencies.[1-3]

The approaches tested in this study are commercially feasible and could be implemented in schools or other institutional food service operations elsewhere. Furthermore, the approaches are applicable to food manufacturers and processors in general. The development of margarines with higher P/S ratios and the replacement of highly saturated coconut, palm, or palm kernel oils with less saturated oils in many food products are examples of such applications. The widespread adoption of such approaches by the food industry could result in healthier foods for Americans.

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Weekly Food Servings and Participation in Social Programs among Low Income Families

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Abstract: Low income families were interviewed to determine factors related to the number of family food servings per week. A multiple regression model indicated that participation in WIC (supplemental food program for women, infants and children), household size, and number of different income sources were associated with more family food servings per week. Number of food servings per week decreased the last week of the month most often in families with younger members. (*Am J Public Health* 1990; 80:1376–1378.)

Introduction

Several studies suggest that family food shortages have increased during the past decade. Populations in the United States that are at risk for these shortages include the elderly, single parent families, and children.[1–10] Other studies have shown that participants of government assistance programs also endure food shortages.[11–16] These food shortages follow monthly cycles with the greatest prevalence occurring the last week of the month.[12]

The purpose of this study was to determine if the number of food servings per week could be used to assess food shortages that occur at the end of the month, the number and types of food and income assistance programs used by low income families, and factors associated with the number of family food servings.

Methods

A random sample of 25 percent of family members from six food cooperatives for low income families was selected, with 109 of 114 responding. Fifty families from the Hillsborough County Florida Expanded Food and Nutrition Education Program (EFNEP) who were not participating in the food cooperative were randomly selected to participate in the study; 12 did not participate and 10 more were randomly selected as substitutes. One home interview was conducted for each family.

The focus of the study was the household. Demographic data were collected with information on participation in 23 food and income assistance programs. The number of family food servings during the past week from a list of 27 food groups was collected. A serving was defined as the preparation and offering of a food group to family members; second and third offerings of the food during the same meal were not counted. A normally distributed additive score for 20 food groups was calculated (mean ± 1 SD: 24.7 ± 5.0; Cronbach's alpha of 0.76.)[17] The excluded food groups were oils, salty snacks, candy, sweet baked goods, soda, soup, and coffee.

Frequency analysis using Chi-square and T-tests for means were used to determine the characteristics of the participants of the two programs. Multiple regression was used to determine factors related to the number of food servings. Potential independent variables for the final model were: participation in individual food and income assistance programs; the number of income or food assistance programs; the reference week of the month for the servings; the household size; sex of respondent; the age of the household respondent; participation in the food cooperatives or EFNEP; and an interaction term between program participation and reference week. However, when the final regression model was constructed several variables were not significant and dropped.

Results

The EFNEP households compared with the food cooperative families were younger: (31 vs 51 years); had more single parents (60 percent vs 40 percent); had more children less than 5 years of age (60 percent vs 44 percent); and had lived less time in their present location (42 vs 128 months).

The food and income sources used by these families are presented in Table 1. There was a wide variation in the combination of sources used among all families with no two families using the same combination; 39 percent had four or more sources and 13 percent had six or seven sources of income.

The last week of the month was associated with a decrease in the number of food servings per week (mean ± 1 SEM 22.9 ± 0.8) compared with the second (26.4 ± 1.0) and third (26.1 ± 0.8) week of the month (Figure 1). The variables in the best model (Table 2) to predict the number of food

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TABLE 1—Food and Income Sources That at Least 10 Percent of the Sample Used[a]

	Food Coop N = 109 %	EFNEP N = 48 %
Food Sources		
Food Pantry	13	10
Commodity	74	33
School Breakfast	32	27
School Lunch	32	33
Food Stamps	48	54
WIC	6	21
Income Sources		
AFDC	24	44
SSI	32	10
Social Security	46	10
Wages	15	27

a) Programs that had less than 10% of participating families included soup kitchens, shelters, senior centers, meals on wheels, credit from stores, retirement funds, veterans, and loans.

TABLE 2—Regression Model to Predict Number of Meals Served in Household during Past Week

	Regression Coefficient	Standard Error
Intercept = 21.7		
WIC[a]	2.3	1.2
Household Size[b]	0.7	0.2
No. Income Sources[c]	1.2	0.4
Week of Month[d]	−0.7	0.4
Week Program[e] Interaction	−0.6	0.1

$F = 14.4$, $r^2 = 0.33$

a) A value of 1 was given to families who participated and a score of 0 for families who did not participate.

b) Household size was a continuous variable.

c) Number of income sources was a continuous variable.

d) Week of the month was a categorical variable that represented the reference week of the month (first to fourth) for the food servings.

e) The interaction term between families from two programs (food cooperative and EFNEP) and the reference week of the month for food servings.

servings per week were: participation in WIC, number of income assistance programs, number of people in the household, week of the month, and an interaction between the week of the month and participation in either the food cooperatives or EFNEP. This interaction represents a greater decrease in the number of food servings the last week of the month in the EFNEP families compared with the food cooperative families.

Discussion

The results from this study support previous reports that end of the month food shortages occur in low income families.[12,18-20] The significant interaction term for the number of food servings identified the EFNEP families who were younger, with children, and less established at their current location as one group at particular risk.

Our study also provides evidence that participation in a greater number of income assistance programs was associated with food servings. However, even after controlling for these factors there was a significant decrease in the number of food servings at the end of the month. This implies that current government support may not be sufficient to last an entire month, especially when the assistance is given one time at the beginning of each month. Several studies of food stamps recipients reported that families use their monthly allotment before the end of the month.* Even families who participate in five different programs have been reported not able to meet the thrifty food plan.[20] Nevertheless, even though there is no guarantee that the diets of families participating in federal programs are of high nutritional quality,[21] assistance programs have been shown to improve the diets of low income families and improve food security.[22]

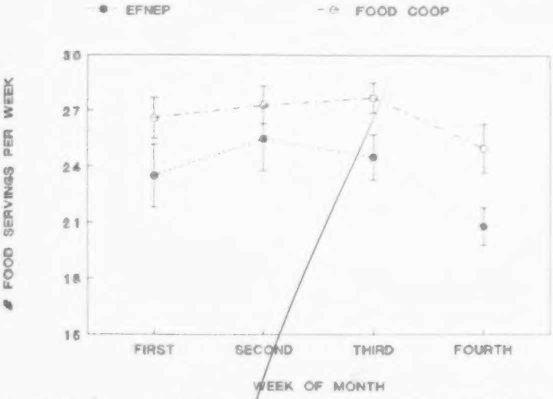

FIGURE 1—Number of Food Servings per Week in Low Income Families (Values represent the adjusted means for the number of servings per week; the vertical bars represent ± 1 standard error.)

In conclusion, weekly food servings decreased the last week of the month for these families and could be measured using a food frequency questionnaire. This pattern may be related to insufficient funds that occur the last week of the month when financial assistance from government sources are depleted.

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The Buddy Volunteer Commitment in AIDS Care

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Abstract: Buddy volunteers provide crucial assistance to people with HIV-related illnesses. Based on volunteers' self-administered questionnaires, our study describes the nature of buddy work. Volunteers indicated their satisfaction with both personal performance and buddy program administration. Several factors were associated with volunteer satisfaction. This report is a first attempt to describe this special relationship created in response to the human immunodeficiency virus (HIV) epidemic. (*Am J Public Health* 1990; 80:1378–1380.)

Introduction

Since the onset of the AIDS (acquired immunodeficiency syndrome) epidemic a decade ago, community-based support for people living with AIDS has increased. Formation of community-based organizations to help people with AIDS (PWAs) has taken place across the United States. Services provided range from food distribution to information hotlines to legal and political advocacy. The size of these organizations ranges from a few dozen active volunteers to several hundred.

This report focuses on programs that establish a special relationship between volunteer and client. "Buddy programs" pair a volunteer from the community with a PWA. The buddy volunteer gives his/her client companionship and basic assistance in living with AIDS, and performs tasks ranging from housekeeping to providing emotional support.[1,2] Thousands of volunteers across America have entered into this profound commitment which often lasts through the death of the person with AIDS.[3]

We attempted to investigate the work done by volunteers at one intermediate-sized AIDS project to determine the nature of the contact existing between the volunteers and the project and between the volunteers and their PWA clients.

Methods

Study Populations

To be eligible for this study, women and men must have completed a buddy training program offered by Rhode Island Project/AIDS prior to February 1989. This process included completion of an application and participation in a multiphase training program. One hundred forty-three past and present buddy volunteers were eligible for the study. To be in the study, active and former volunteers returned a self-administered questionnaire. This report is based on information from the 67 volunteers who completed the questionnaire.

Data Collection

An anonymous, self-administered questionnaire was distributed to all active and inactive buddy volunteers. Initially, questionnaires were coded to be matched with the volunteers' initial applications to the program so comparisons could be made between questionnaire and application data. Buddy support group leaders distributed the questionnaires. Inactive volunteers were mailed a questionnaire, along with a cover letter and a self-addressed stamped envelope, to their last known address.

To improve response, a second batch of questionnaires was distributed to group leaders with instructions to mail them back directly to the investigators. Inactive volunteers

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who had not previously returned a questionnaire were sent another one with a second cover letter.

Data Analysis

Active and inactive populations were examined together. Satisfaction with various aspects of program administration and with personal performance as a buddy volunteer was the conceptual outcome of interest. A satisfaction score was devised to classify respondents as satisfied, dissatisfied, or indeterminate. To quantify an individual's satisfaction, we created a five-point scale relating to a volunteer's levels of satisfaction with program administration and personal performance as a buddy; among these items were queries into the nature and perception of typical requests clients made to buddy volunteers, such as help with cleaning, transportation, or meal preparation. Other questionnaire items were examined in relation to this scaled outcome.

Most differences between the two groups were observed through the comparison of proportions. Satisfaction prevalence ratios were examined for certain determinants, namely lag time between becoming a buddy and being assigned a client, number of the client's total requests for specific buddy services per week, and volunteer's assessment of client's supportive relationships. Confidence intervals for these measures were calculated using Miettinen's test-based procedure.[4]

To evaluate whether the segment of the buddy volunteer population responding to the questionnaire was representative of the buddy volunteer population as a whole, data from the applications of buddies known to have responded to the questionnaire (in the first mailing) were compared to data collected from applications of the nonrespondents.

Results

Volunteer and Client Profiles

Fifty-three percent of buddies responding to the questionnaire were male, and most volunteers (73 percent) were 26 to 45 years old. Of 61 buddies identifying with a specified race category, only five (8 percent) reported themselves as other than White. When compared with AIDS cases in Rhode Island or with the client population, minorities were underrepresented among the volunteers. According to Rhode Island Project/AIDS, their buddy program serves 63 percent of the state's IV (intravenous)-drug-related AIDS cases.

The Buddy Experience

Tables 1 and 2 show distributions of experiences as buddy volunteers: Table 2 focuses on the weekly requests that are made to volunteers from their clients; Table 1 shows most buddies responding had been assigned at least one client; over 20 percent had been assigned two or more. Over one-third of buddies had experienced the death of a client. Few buddies had requested reassignment while their clients were alive. Over half of buddies reported that being a buddy had increased the stress in their lives.

The most significant problems faced in working with PWAs were the time commitment and emotional investment (Table 1). Dissatisfied buddies reported more requests for certain kinds of tasks than satisfied volunteers, such as cleaning and preparing meals (Table 2). Almost all the buddy volunteers visited their client at least once weekly. Over half provided transportation at least once per week. Shopping, cleaning, and preparing meals were done less often. Table 3 shows a distribution of the hours spent per week by buddy volunteers according to satisfaction status. Over 60 percent of volunteers spent six or more hours weekly providing buddy services.

TABLE 1—Distributions of Factors in Buddy Experience According to Satisfaction Status [number reporting factor/number reporting satisfaction status (percent)*]

	Satisfied N (%)	Dissatisfied N (%)	Overall*** N (%)
Total Clients			
none	1/20 (5)	6/36 (17)	12/65 (18)
one	12/20 (60)	23/36 (64)	39/65 (60)
two	6/20 (30)	6/36 (17)	12/65 (18)
three	1/20 (5)	1/36 (3)	2/65 (3)
Total Deceased Clients			
none	12/20 (60)	23/36 (64)	41/64 (64)
one	6/20 (30)	10/36 (28)	18/64 (28)
two	2/20 (5)	3/36 (8)	5/64 (8)
Requests for Reassignment	1/20 (5)	3/36 (8)	6/66 (9)
Major Problem Cited			
time commitment	**	12/36 (33)	22/55 (40)
emotional investment	**	8/36 (22)	15/55 (27)
other	**	12/36 (33)	18/55 (33)
Stress Change Since Becoming a Buddy			
increased	12/20 (60)	18/33 (55)	36/66 (55)
decreased	3/20 (15)	4/33 (12)	17/66 (28)
same	5/20 (25)	11/33 (33)	13/66 (20)

*All percents are rounded to the nearest whole number; totals may not equal 100%.

**None of the "satisfied" respondents reported a major problem.

***"Overall" includes all respondents to the question: "satisfied," "dissatisfied," and intermediate.

TABLE 2—Frequency of Weekly Requests According to Satisfaction Status [number reporting request/number of respondents in satisfaction category (percent)*]

Number of Requests/Week	Satisfied N (%)	Dissatisfied N (%)	Overall** N (%)
Personal Visits			
0	1/19 (5)	1/28 (3)	2/52 (4)
1	7/19 (37)	8/29 (28)	18/52 (35)
2 or more	11/19 (58)	20/29 (69)	32/52 (62)
Shopping			
0	11/19 (58)	15/29 (52)	44/49 (90)
1	6/19 (32)	10/29 (35)	17/51 (33)
2 or more	2/19 (11)	4/29 (14)	6/51 (12)
Cleaning			
0	17/18 (95)	24/28 (86)	44/49 (90)
1	1/18 (6)	3/38 (11)	5/49 (10)
2 or more	0/18 (0)	1/28 (4)	1/49 (2)
Preparing Meals			
0	16/18 (89)	22/28 (79)	41/49 (90)
1	2/18 (11)	3/28 (11)	5/49 (10)
2 or more	0/18 (0)	3/28 (11)	3/49 (6)
Transportation			
0	7/19 (37)	13/29 (45)	22/52 (42)
1	2/28 (11)	7/29 (24)	16/52 (31)
2 or more	4/19 (21)	9/29 (31)	14/52 (27)
Telephone Calls			
0	2/18 (11)	3/29 (10)	6/51 (12)
1	5/18 (28)	7/29 (24)	13/51 (26)
2 or more	11/18 (61)	19/29 (66)	32/51 (63)

*All percents are rounded to the nearest whole number; totals may not equal 100%.

**"Overall" includes all respondents to the question: "satisfied," "dissatisfied," and intermediate.

Buddy volunteers were twice as likely to report feeling satisfied if assigned to their first PWA one to five months after acceptance to the Buddy Program at Rhode Island Project/AIDS (prevalence ratio = 2.0; 95% confidence interval = 0.8,

TABLE 3—Distribution of Total Hours Devoted to Buddy Work by Satisfaction Status [number in category/number of respondents (percent)*]

# Hours per Week Devoted	Satisfied N (%)	Dissatisfied N (%)	Total N (%)
≤5 hours/week	7/19 (36)	12/31 (39)	19/50 (38)
6–10 hours/week	6/19 (32)	6/31 (19)	12/50 (24)
≥11 hours/week	6/19 (32)	13/31 (42)	19/50 (38)

*All percents are rounded to the nearest whole number; totals may not equal 100%.

5.1). Volunteers with 10 or less requests per week from their clients also reported greater satisfaction (prevalence ratio = 3.2; 95% ci = 1.5, 6.0). Buddies serving PWAs with more supportive relationships (other than buddy-client) in their lives were twice as likely to be satisfied in their work (prevalence ratio = 2.0; 95% ci = 0.8, 4.7).

Discussion

This study may be the first attempt to describe the work of AIDS volunteer programs such as the buddy program of Rhode Island Project/AIDS. Comparison of buddy/client and client/state AIDS case demographics suggest that minorities are underrepresented in these volunteer populations. While none of the volunteers responding to our questionnaire reported themselves as representing the Latino population, Rhode Island Project/AIDS' proportion of Black and Latino clients (25 percent)[5] is comparable with the state's proportion of Black and Latino cases (29 percent).[6]

This study describes the variety of services performed by buddy volunteers, and their interaction with the agency. Although there was considerable comparability between respondents and nonrespondents with respect to motivation for volunteering and providing support as buddies, we are cautious about generalizing the survey findings because the response rate was low.

The Buddy Program is a valuable, volunteer-based health care service that is critically needed by people with HIV (human immunodeficiency virus)-related illnesses. This description may stimulate increased appreciation of this service as well as promote an understanding of how to organize volunteers' efforts and support their needs.

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Epidemiology of Reported Cases of AIDS in Lesbians, United States 1980–89

Susan Y. Chu, PhD, James W. Buehler, MD, Patricia L. Fleming, PhD, and Ruth L. Berkelman, MD

Abstract: National surveillance data for reported cases of acquired immunodeficiency syndrome (AIDS) were used to assess demographic characteristics and behavioral risk factors in lesbians. From June 1, 1980 through September 30, 1989, 79 women with AIDS reported sex relations only with a female partner; most of these women (95 percent) were intravenous drug users. Prevention of human immunodeficiency virus (HIV) infection in the lesbian community will require efforts to prevent and reduce intravenous drug use. (*Am J Public Health* 1990; 80:1380–1381.)

Introduction

Very little has been written on the epidemiology of acquired immunodeficiency syndrome (AIDS) and human immunodeficiency virus (HIV) infection in the lesbian population. Two instances of female-to-female sexual transmission of HIV have been reported,[1,2] and there is some concern that the risk of infection in female homosexuals may be underestimated because of the lack of information about this population group.[3] This report describes various demographic characteristics and behavioral risk factors of lesbians with AIDS in the United States.

Methods

We used national surveillance data for 9,717 cases of AIDS in adult women reported to the Centers for Disease Control (CDC) between June 1, 1980 and September 30, 1989. Only those that met the CDC surveillance case definition for AIDS were included in our analysis.[4] AIDS patients were grouped according to their reported sexual behavior, using responses to the following two questions:

- "After 1977 and preceding the diagnosis of AIDS, did this patient have sexual relations with a male partner?"
- "After 1977 and preceding the diagnosis of AIDS, did this patient have sexual relations with a female partner?"

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Women who had sexual relations only with female partners were classified as lesbian, women who had both male and female sexual partners were classified as bisexual, and women who had only male sexual partners were classified as heterosexual. For women who were classified as lesbian, recorded data (e.g., sex, sex partners, mode of transmission) were confirmed directly with the reporting health departments to minimize misclassification and data entry errors. We excluded 1,242 women who could not be classified as lesbian, bisexual, or heterosexual because of incomplete responses to the above two questions. However, 90 percent of these women had a known risk for HIV exposure (intravenous drug use, heterosexual contact with a person at increased risk for, or known to be, infected with HIV, or blood transfusion).

Persons with AIDS were classified in a hierarchy of exposure categories according to their presumed means of acquiring HIV infection. Those with more than one possible means of acquisition were classified only in the exposure listed first in the hierarchy, by using the following sequence: 1) intravenous drug use, 2) receipt of blood products, 3) heterosexual contact with an HIV-infected partner (or having a specified risk for HIV), and 4) "no identified risk." The last category represents persons who had none of the other risk exposures above, and therefore could include women who acquired HIV infection through female-to-female sexual contact.

Results

As of September 30, 1989, there were 79 reported cases of AIDS in lesbians, representing 0.8 percent of all reported adult women with AIDS in the United States.

As shown in Table 1, most of the lesbians with AIDS (95 percent) were intravenous drug users (IVDUs). The remaining 5 percent acquired HIV infection through the receipt of blood or blood products. As with IVDU-associated AIDS in all women, most lesbian women with AIDS were younger (85 percent were less than 40 years old), Black or Hispanic (80 percent), and resided in the northeast (77 percent). There were 103 reported cases of AIDS in bisexual women. Most bisexual women with AIDS also were IVDUs (79 percent), 16 percent had sex partners at increased risk or known to be infected with HIV, and 4 percent had histories of blood transfusion.

We also compared the occurrence of reported opportunistic diseases among homosexual, bisexual, and heterosexual women with AIDS. The only notable difference was in the occurrence of Kaposi's sarcoma (KS). KS (including presumptive and confirmed diagnoses) was not reported in any of the homosexual females with AIDS but was reported in 9 percent (9/103) of bisexual females and 2 percent (167/8,293) of heterosexual females with AIDS.

TABLE 1—AIDS cases in Lesbians (June 1980-September 1989) reported in the United States

AIDS Cases	No. (%)
Exposure category	
IVDU	75 (95)
Transfusion	4 (5)
Age (years)	
13–19	0 (0)
20–29	19 (24)
30–39	48 (61)
≥40	12 (15)
Race/ethnicity	
White	15 (19)
Black	30 (38)
Hispanic	33 (42)
Other	1 (1)
Region	
Northeast	61 (77)
Midwest	0 (0)
South	7 (9)
West	3 (4)
Territories	8 (10)

Discussion

Lesbians represent a very small proportion of women with AIDS in the United States, and most cases are related to intravenous drug use. Prevention of HIV infection in the lesbian community will require efforts to prevent and reduce IV-drug use—the major and most direct means of transmission in this population.

Although there is no specific exposure category for female-to-female transmission, lesbians who acquired HIV infection from female-to-female contact would have been classified in the "no identified risk" (NIR) exposure category, of which there were none. Female-to-female transmission of HIV also could occur among bisexual females; however, only one bisexual female had no identifiable risk and was classified in the NIR category.

Although it is not possible to determine whether differences in the occurrence of Kaposi's sarcoma (KS) reflect differences in exposures, we also observed that among women with AIDS who reported sex with a *bisexual* man, 4.1 percent (20/493) had KS reported, whereas among women who did not report sex with a *bisexual* man, 1.6 percent (52/3162) reported KS. Moreover, the frequencies of reported KS were higher among homosexual (22 percent) or bisexual men (14 percent) than among heterosexual men (3 percent) (whose predominant means of HIV exposure was intravenous drug use). These patterns are compatible with the hypothesis that KS is a sexually transmitted infection that occurs in association with HIV infection and disproportionately affects homosexual and bisexual men.[5]

It is important to note that we used categories based on reported sexual behaviors, not on self-labeled sexual orientation. The labels "heterosexual" and "homosexual" do not always predict sexual behavior.[6] Our "lesbian" category does not necessarily represent women who identify themselves as lesbians, but more accurately represents women with AIDS who reported having sexual relations only with a female partner since 1977.

Although female-to-female transmission of HIV appears to be an extremely rare event, the occurrence of AIDS among lesbian and bisexual women indicates that women who engage in sex with other women can be exposed to HIV. Case reports of female-to-female sexual transmission of HIV[1,2] and the well-documented risk of female-to-male HIV transmission indicate that vaginal secretions and menstrual blood are potentially infectious and that mucus membrane (e.g., oral, vaginal) exposure to these secretions can potentially lead to HIV transmission.

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Prior Cesarean Delivery in Women with Secondary Tubal Infertility

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Abstract: The history of cesarean delivery was evaluated in a population-based case-control study of secondary infertility in King County, Washington. Sixty-one married women diagnosed with secondary infertility due to tubal problems who had a previous viable pregnancy were compared to 343 married women who had a previous viable pregnancy and then had a live birth that was conceived at the same time the infertile women began trying to conceive. The risk of tubal infertility was not substantially elevated in women who had a previous cesarean delivery in the most recent viable pregnancy compared to women with vaginal delivery (odds ratio = 1.2; 95% confidence interval = 0.4, 3.7). (*Am J Public Health* 1990; 80:1382–1383.)

Introduction

Cesarean section deliveries in the United States have increased from 5.5 per 100 deliveries in 1970 to 20.3 in 1983.[1,2] Women who have cesarean deliveries are at greater risk for postpartum complications of pelvic infections than women who deliver vaginally,[3] but the effect of cesarean delivery and related postpartum infection on subsequent fertility is not well understood.[4–10]

Methods

A case-control study design was used, in which the cases were 61 20–39 year old married women residents of King County, Washington who had secondary infertility (women who had previously conceived, but who were not able to conceive after trying for at least one year) and who have previously delivered a live birth or still birth. These women had their first medical evaluation for infertility during 1979 to 1981. (The study subjects were part of a larger study on infertility and contraception in which 89.5 percent of all physicians in King County who perform infertility evaluations agreed to participate.)[11] If cesarean section via a possible infection did lead to infertility in some women, it would more likely be women whose infertility was related to a tubal condition.[12,13] For this reason only women with blocked tubes, diagnosed by hysterosalpingogram or surgery, were included as cases. Women with an abnormal hysterosalpingogram who were later found by surgery to have an intrauterine abnormality, rather than a tubal condition, were excluded.

The control group (n = 343), identified from state vital records, was composed of 20–39 year old married women residents of King County who had a previous birth and then subsequently delivered a newborn who was conceived during the same years in which the infertile cases started trying to become pregnant.

By structured interview, data for each study subject were collected on demographic characteristics, use of "social" drugs, tobacco, alcohol, personal hygiene, and medical, reproductive, contraceptive, and sexual histories.

Results

Although cases and controls were somewhat similar with regard to education, income, and age at reference date (the time at which both the infertile case and the control began to attempt to conceive), the cases had a larger percentage of racial minorities and were more likely to have been employed, to be a smoker, and to have had more sexual partners, a history of Dalkon Shield use, and their last viable pregnancy before 1975 than did the controls (Table 1).

A cesarean section delivery of the most recent birth was found in four cases (6.6 percent) and in 29 controls (8.3 percent). Using unconditional logistic regression to obtain maximum likelihood estimates of relative risk, we adjusted for the effects of age (less than 30, 30 or older) and year of last viable pregnancy (before 1975, 1975 or after). The adjusted odds ratio, an estimate of the relative risk, was 1.2 (95 percent confidence interval = 0.4, 3.7). Since the number of exposed cases was small, we also performed exact stratified analysis with EGRET statistical software which utilizes the method described by Mehta, Patel and Gray.[14] The exact odds ratio, adjusted for the same variables as above had slightly wider confidence intervals (OR = 1.2; 95% CI = 0.3, 3.8). Variables that were assessed but did not appreciably change the cesarean delivery risk estimates included race, employment type, smoking status, parity, education, income, age at first coitus, number of lifetime sexual partners, history of Dalkon Shield and other intrauterine device use, history of gonorrhea, pelvic inflammatory disease, douching, marijuana use, and census tract of residence.

In our study, no case or control with a cesarean delivery reported postpartum pelvic infection.

Discussion

Although this study revealed no association between cesarean delivery and subsequent secondary tubal infertility, the small sample size limited the study's ability to detect a possible modest increase in risk associated with cesarean delivery. The 95% CI suggests a relative risk of 3.7 or more is highly unlikely.

Another limitation of the data is that women with a prior successful pregnancy who had a cesarean delivery may be less motivated to seek help or to undergo the expense or discomfort of invasive techniques, i.e. hysterosalpingogram or laparoscopic surgery necessary to diagnose tubal infertility. To the extent this is so, ascertainment of cases of women with secondary tubal infertility among women with cesarean section will be incomplete, and the observed risk will underestimate the true risk.

Finally, we were unable to assess the more direct measure of post-cesarean pelvic infection itself, except by self-report. Medical charts were not reviewed.

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Table 1—Characteristics of Women with Secondary Tubal Infertility and Controls

Characteristic (at reference date)	Cases		Controls	
	No.	(%)	No.	(%)
Education				
<College	24	(39.3)	133	(38.8)
College	37	(60.7)	210	(61.2)
Annual Income[a]				
<$15,000	10	(16.9)	56	(16.4)
$15,000–30,000	30	(50.8)	180	(52.8)
>$30,000	19	(32.2)	105	(30.8)
Age Distribution (years)				
<20	3	(4.9)	1	(0.3)
20–24	9	(14.8)	56	(16.3)
25–29	32	(52.5)	159	(46.4)
30–34	14	(23.0)	110	(32.1)
35+	3	(4.9)	17	(5.0)
Race				
White	48	(78.7)	310	(90.4)
Black	7	(11.5)	10	(2.9)
Other	6	(9.8)	23	(6.7)
Employment Type				
None	16	(26.2)	177	(51.6)
Executive	3	(4.9)	9	(2.6)
White Collar	27	(44.3)	128	(37.3)
Blue Collar	15	(24.6)	29	(8.5)
Smoking Status				
Current	26	(42.6)	88	(25.7)
Never	28	(45.9)	192	(56.0)
Prior	7	(11.5)	63	(18.4)
Total Number of Lifetime Sexual Partners[b]				
1–4	39	(63.9)	263	(76.9)
≥5	22	(36.1)	79	(23.1)
Prior Dalkon Shield Use				
Yes	12	(19.7)	30	(8.7)
No	49	(80.3)	313	(91.3)
Reference Year				
<1976	8	(13.1)	21	(6.1)
1976–1978	25	(41.0)	128	(37.3)
≥1979	28	(45.9)	194	(56.6)
Parity				
1	50	(82.0)	266	(77.6)
2	7	(11.5)	55	(16.0)
3	4	(6.5)	14	(4.1)
≥4	0		8	(2.4)
Year of last viable pregnancy				
Before 1975	38	(62.3)	91	(26.5)
1975 or after	23	(37.7)	252	(73.5)

[a] Two cases and two controls did not answer this question.
[b] One control refused to answer.

Despite these limitations, this study adds to our knowledge; it is the first reported population-based case-control study which assesses the relative risk of secondary infertility following cesarean section. Our findings corroborate those of Hemminki, *et al*,[4] and Zdeb, *et al*,[6] who found essentially no difference in the proportion of women who had at least one subsequent child after the first pregnancy when comparing women with previous cesarean and vaginal deliveries.

The findings are in contrast to those found by La Sala[7] and Hemminki.[5] Of all the reported studies, only La Sala adequately excluded women who had been sterilized, were using contraceptives, or were not attempting a pregnancy. However, La Sala defined secondary infertility as two years of infertility, a definition not widely used in the United States.

Three other studies that addressed this topic found similar rates of subsequent pregnancies in women with emergency cesareans[9] or cesareans with subsequent infection[8,10] compared to other women with elective cesarean or cesarean without infection respectively.

One explanation for why a large increase in risk of infertility was not found in our study may be prophylaxis or early diagnosis and treatment of postpartum infections in women with cesarean deliveries which could prevent the severity and course of infection and thus decrease the occurrence of tubal damage.

It will take larger studies or several studies to be confident in these results. It is nonetheless reassuring that in a metropolitan area of nearly 1.3 million people in which most of the incident cases of infertility who sought help were identified, only four of the 61 cases of secondary tubal infertility with a prior live birth had a cesarean section in the last delivery, a rate similar to the comparison group and to obstetric patients seen during the same time period.

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A Comparison of Homeless, Community-wide, and Selected Distressed Samples on the CES-Depression Scale

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Abstract: Center for Epidemiological Studies (CES) Depression Scale results for surveys of homeless, community-wide and selected distressed samples are compared. Nearly four times the percentage of homeless fit the criterion for clinical caseness (a score of 16+) compared to the general population (74 to 19 percent). None of the distressed samples exhibited a higher rate except psychiatric patients diagnosed as acutely depressive. High rates of depression have implications for social policies directed toward homelessness. (*Am J Public Health* 1990;80:1384–1386.)

Introduction

Psychological depression is characterized by depressed mood, negative self-concept, disturbed vegetative functioning, agitation, slower activity levels, distractibility, and indecisiveness.[1] These symptoms of extreme distress are especially detrimental for problem solving and coping among homeless persons with few financial and social resources, and limited control over their environments.

The Center for Epidemiological Studies Depression Scale (CES-D)[1–3] is a survey instrument scored from 0 to 60 (20 symptoms coded 0 to 3 depending on their frequency during the week prior to interview). It has high internal consistency,[1] strong test-retest reliability[4] and, although designed to measure depressive symptomatology in community populations, it is a sensitive predictor of diagnosed depression.[4–7] For example, Boyd, *et al,* found 80 percent correspondence between diagnosed depression and "possible clinical caseness" (a CES-D score of 16+).[8] The scale's specificity is strong; over 91 percent were correctly predicted to have nondepressive diagnosis (using the Diagnostic Interview Schedule) when the 21+ cutoff was used for the CES-D.[4] For our homeless sample, the Cronbach's alpha reliability coefficient for the CES-D was .89; item-to-total correlations ranged from .24 to .74.

Using the CES-D Scale,[1–3] we compared rates of depressive symptoms for a sample of homeless persons to existing data on community-wide and selected distressed samples.

Methods

Data on the homeless were from the Birmingham (Alabama) Homeless Enumeration and Survey Project conducted in early 1987.[9] The Metropolitan Statistical Area (MSA) population was approximately 900,000. The enumeration phase was a single-night census of homeless persons finding 498 in MSA shelters, and 103 on the streets downtown where the homeless were known to be concentrated. An interview survey of 150 adults followed in the three months after the enumeration. A random probability sampling design was used with quotas for the variables sex, race, and geographic site (i.e., shelter versus street) based on enumeration data. Whites were slightly overrepresented in the street sample. Interviews averaged 62 minutes. Of 155 people asked for interviews, only one refused. Four interviews were unusable.

Results

Table 1 presents percentages of possible clinical caseness (CES-D of 16+) for community-wide, selected distressed, and homeless samples.* Across five general population studies in various communities between 1971 and 1985, the results fell consistently below 20 percent. In the distressed samples (discounting acute depressives), percentages ranged from 23.0 (physically or psychologically impaired persons over 55) to 79.0 (rural mental health clinic patients). Generally, the distressed samples comprised of psychiatric patients had higher rates than samples characterized by stressful life circumstances.

Comparing community-wide and distressed samples to our sample revealed a considerable amount of distress among the homeless. Only one respondent of our sample was asymptomatic. Nearly three of four (73.3 percent) scored 16 or above. This percentage was similar to Koegel and Burnam's[14] finding of 71 percent clinical caseness among homeless males of inner-city Los Angeles. In addition, we found 37 percent to score 30 or above, a result similar to Susser's 33 percent for a sample of male first-time shelter users primarily under 40 years of age.[19] Except for Weissman's study of clinic patients diagnosed with acute depression,[6] even groups as distressed as psychiatric inpatients, alcoholics under treatment, and rural mental health clinic patients did not have significantly higher rates.

Comparisons to domiciled samples with stressful life circumstances were informative. For example, low income mothers of young children had significantly lower rates than the Birmingham homeless, as did the unemployed, suggesting that environmental and situational stressors of homelessness exact a greater toll on mental well-being than those faced by the housed poor. Finally, for persons recently widowed or separated—two highly stressful life events—the rates of clinical caseness were lower than that of the homeless sample. An analysis elsewhere of variation in depressive symptomatology within our homeless sample revealed that homelessness does not impact its victims differentially.[20] No matter how the sample was broken down, the mean CES-D score for any subsample was more than twice the mean score of any of the community-wide samples.**

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*Analysis of mean differences in CES-D scores yielded the same results. These data are available upon request from the authors.

**A table summarizing mean differences of sample subgroups is available upon request from the authors.

TABLE 1—Comparison of CES-D Scores: Community-Wide, Selected Distressed, and Homeless Samples

Studies and Year(s)	Research Site/Sample/Subsamples	N	Percent CES-D 16 or above
Community-Wide Samples			
Radloff;[1] Comstock & Helsing[5] (1971–73)	Washington County, Maryland & Kansas City, Missouri	2845	19.5**
Eaton & Kessler;[10] Sayetta & Johnson[11] (1971–75)	National Health & Nutrition Examination Survey	2867	16.4**
Frerichs, et al.[12] (1979)	Multi-Ethnic sample, Los Angeles	1000	19.1**
Ensel;[4] Lin and Ensel[13] (1979–80)	Tri-County Area, Upstate New York	871	16.9**
Koegel and Burnham[14] (1984–85)	Inner-City Los Angeles, Males only	1466	9.4**
Selected Distressed Samples			
Radloff[15] (1971–73)	Washington County, Maryland & Kansas City, Missouri		
	Psychiatric Inpatients	70	70.0
	Death of spouse-past year	33	51.5*
	Marital separation-past year	63	49.2**
	Unemployed	199	30.2**
Weissman, et al.[6] (1977)	Connecticut Mental Health Center, New Haven		
	Acute Depressives	148	99.3**
	Recovered Depressives	87	43.7**
	Drug Addicts	60	51.7**
	Alcoholics	61	62.3
	Schizophrenics	50	36.0**
Hall, et al.[16] (1976–77)	Greensboro, North Carolina; Low-Income Mothers of Young Children	111	48.0**
Husaini, et al.[17] (1977)	9 Rural Counties, Tenn.; Mental Clinics and Veterans Administration Hospital	194	79.0
Orr, et al.[7] (1984)	Baltimore; Mothers of Young Children	149	38.3**
Frerichs, et al.[12] (1979)	Multi-Ethnic sample, Los Angeles		
	Unemployed	98	46.9**
	Low Total Family Income ($0–$8,499)	239	29.3**
Another authors' sample[a] (1986–87)	4 Metropolitan Counties in Alabama: Physically and/or psychologically impaired adults, 55 years and older	154	23.0**
Homeless Samples			
Koegel and Burnam[14] (1984–85)	Inner-City Los Angeles, Males only	379	71.0
Rossi, et al.[18] (1987)	Chicago	319	47.0[b]
Present study (1987)	Birmingham, Alabama	150	73.3

[a] FROM: La Gory M, Fitzpatrick KM: The effects of environmental context on elderly depression. Presented to the American Gerontological Society Meetings, Minneapolis, Minnesota, 1989.
[b] These figures cannot be compared directly with others. Only six CES-D items were used.
*Significantly different from Birmingham homeless, $p < .05$, two-tail test.
**Significantly different from Birmingham homeless, $p < .001$, two-tail test.

Discussion

The homeless suffer from high rates of depressive symptomatology indicative of extreme distress. Only acute depressives, mental health clinic patients, psychiatric inpatients, and alcoholics have rates of clinical caseness on the CES-D comparable to the homeless. Some caveats are in order. First, given the extreme environmental conditions of homelessness (e.g., poor nutrition, climatic exposure), the CES-D may be tapping physiological as well as psychological dimensions.[6] Second, the CES-D may be, for some respondents, gauging reactions to acute situational factors rather than gauging depressive illness or chronic demoralization.[13,21–24] Third, homelessness is usually accompanied by other stressful life events and circumstances; thus, depression may precede homelessness in some cases.[24] Notwithstanding these considerations, depressive symptoms have implications for an individual's ability to resolve the situation, even if external conditions (e.g., available jobs and housing) are favorable. These results suggest that current shelter, rehousing, and treatment programs should institute intervention strategies to avert the potentially cyclical nature of homelessness and depression.

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How Valid Are Mammography Self-Reports?

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Abstract: We compared mammography reports in medical records to self-reports obtained during a 1989 telephone interview survey for a sample of 100 women members of a health maintenance organization (HMO) who indicated they had mammograms within the past year and 100 who said they had not had mammograms within the past year. Of the women reporting they had not had mammograms within the past year, none had mammogram reports in the HMO data center. Of the 100 women reporting they had mammograms within the past year, 94 had confirmatory radiology records. (*Am J Public Health* 1990; 80:1386–1388.)

Introduction

Programs of regular mammography screening can produce downward shifts in the stage of breast cancer and reduce mortality by as much as 35 percent.[1–4] The National Cancer Institute (NCI), the American Cancer Society, and other major medical organizations recommend annual screening mammograms for all women ages 50 and older and screening every one to two years for women ages 40–49 years.[5] A potential problem in evaluating the effectiveness of programs aimed at increasing mammography utilization is that most surveys and program evaluations have relied exclusively upon women's self-reports of mammograms. The draft of the US Department of Health and Human Services' Year 2000 Objectives for the Nation stipulates that one of the key assumptions underlying the tracking of changes in mammography utilization is that self-report data provide a valid measure of screening utilization.[6]

If mammography self-reports are not accurate, survey data may overestimate or underestimate actual mammography utilization. Although the accuracy of self-report data in such diverse areas as cervical cancer screening and smoking cessation has been studied,[7–11] the findings cannot be generalized from one content area to another.[7] Based on a MEDLINE search, we found no published studies in which mammography self-reports were validated. Therefore, we undertook the present study.

Methods

This validation study was part of the Avoidable Mortality Study,[4] conducted in conjunction with US Healthcare Check, a free breast screening program offered to all women members of an IPA-model (independent practice association) HMO (health maintenance organization) ages 40 years and over. National guidelines are followed: mammograms are recommended every one to two years for women ages 40 to 49 years and annually for women age 50 and older.[5]

Four telephone survey waves are conducted as part of the Avoidable Mortality Study to evaluate the effectiveness of the program in increasing mammography utilization among women in the HMO compared to geographic controls. US Healthcare Check maintains a centralized mammogram report database. Radiology reports of HMO members are sent to the US Healthcare Check office and to the women's physicians.

In January 1989, a telephone survey was conducted as part of the Avoidable Mortality Study. A sample of 450 HMO women between the ages of 50 and 74 was selected randomly from approximately 45,000 eligible women to whom the US Healthcare Check program is targeted. Interviews were completed with 84 percent; fewer than 10 percent of the women refused to be interviewed.

During the 15-minute structured telephone interview conducted by professional interviewers, women who said they had mammograms were asked when the procedure was most recently done. Mammography utilization questions were those used in the National Health Interview Survey (NHIS) 1987 Cancer Control Supplement and by members of the NCI's Breast Screening Consortium.[4] Overall, 63 percent of the HMO respondents said they had a mammogram in the past year. Mammography rates similar to those in our study and higher than the NHIS[12] also were found in surveys

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conducted by five other NCI Mammography Consortium sites.[13] We questioned whether overreporting could account for some of these differences.

In August 1989, we randomly selected 100 women from the 278 HMO women whose responses indicated they *had* a mammogram within the past year and 100 HMO women from the 171 who indicated they *had not had* a mammogram within the past year. These women comprised the validation study sample.

For women reporting having had mammograms within the past year, project staff contacted the HMO assigned radiology center if the database contained no confirmatory record. If the center was unable to verify the woman's mammogram through radiology reports, the primary physician was identified through the database and contacted. If the physician had no record of the mammogram, the woman herself was contacted to provide additional information about where and when she had the mammogram. Subsequently, with the woman's consent, study staff contacted the radiology center indicated for confirmation of the mammogram. In all instances, women's self-reports were considered valid only if they reported having had a mammogram, and this report was validated by database information or confirmed by radiology or physician's office records.

Results

Of the 200 women originally selected for the validation study, one had to be eliminated due to a data entry error. In general, women reporting having had a mammogram within the past 12 months did not differ from those reporting not having had a mammogram within the past 12 months. However, working women were significantly less likely to have obtained mammograms, and there were more employed women in the group who had not had mammograms (Table 1).

Women's self-reports of having obtained mammograms within the past year correlated highly with the presence of actual mammogram reports (Table 2). Of the 99 women reporting they had *not* had mammograms within the past year, none had a mammogram report in the database. Of the 100 women reporting they "had a mammogram" within the past year, we were able to validate self-reports on 94 women. Mammogram reports on 50 of these women were obtained through the database; the remaining 44 were validated through phone calls to radiology centers or to the women's physicians. Although *all* of the remaining six women had obtained mammograms, they were not done within the past year. Five of the women had mammograms between 13 and 18 months prior to the survey interview; the other woman had a mammogram two years prior to the survey. These six women did not share any particular sociodemographic characteristics when examined by age, employment, race, education, and marital status. Thus, ultimately, we were able to account for *every* one of the women who reported that they had mammograms.

Discussion

These data indicate agreement between women's self-reports of mammography and providers' records. But, for the 99 women reporting they had *not* had mammograms within the past year, we did not attempt further verification beyond the data in the US Healthcare Check database, as we did for the women reporting they had obtained mammograms within the past year.

TABLE 1—Demographic Characteristics of Women in the Mammogram Validation Study

	Mammogram in last 12 months				
	Yes		No		
Characteristics	n	(%)	n	(%)	*p*
Age (years)					
50–54	24	(24)	29	(29)	
55–59	30	(30)	32	(33)	
60–64	16	(16)	20	(20)	
65–69	19	(19)	11	(11)	
70–74	11	(11)	7	(7)	
Total	100	(100)	99	(100)	p = .40
Race					
White	80	(80)	80	(81)	
Non-White	20	(20)	19	(19)	
Total	100	(100)	99	(100)	p = .80
Annual Income					
<$15,000	22	(28)	19	(22)	
$15,000–$30,000	31	(39)	32	(38)	
>$30,000	20	(33)	34	(40)	
Total	79*	(100)	85*	(100)	p = .58
Marital Status					
Married	72	(73)	62	(64)	
Not Married	26	(27)	36	(37)	
Total	98*	(100)	98*	(100)	p = .14
Education					
<High School Graduate	21	(21)	25	(25)	
High School Graduate	50	(51)	45	(46)	
>High School Graduate	28	(28)	28	(28)	
Total	99*	(100)	98*	(100)	p = .77
Employment					
Employed	57	(58)	74	(76)	
Not employed	42	(42)	24	(24)	
Total	99*	(100)	98*	(100)	p = .01

*Numbers do not equal total n due to refusals or missing data.

It is not feasible to verify such negative reports beyond the HMO database, since it would require contacting every physician who could have ordered a mammogram for each woman. At first, only 50 percent of women who reported having a mammogram had verification in the HMO database. These omissions were due to the radiologist failing to report the procedure in a timely fashion; the women had responded correctly. For a similar rate of omission to exist among the women reporting *not* having mammograms would also require an error in self-report on the part of the majority of women. Given the high rate of accuracy among women reporting mammograms, a high rate of inaccurate response among women reporting no mammogram seems highly unlikely.

In a study by Sawyer, *et al*,[11] of the accuracy of women's self-reports of Pap tests, the underreporting errors—although less than the overreporting errors of 21 percent—were 17

TABLE 2—Comparison of Women's Self-reports with Actual Mammogram Reports

	Radiology Reports		
Self-reports	Yes n	No n	Total n
Had mammogram in preceding year	94	6	100
Did not have mammogram in preceding year	0	99	99
Total	94	105	199

Phi coefficient = .94 (95% CI = 0.93, 9.95), p < .001.

percent. Unlike having a Pap test, which is usually taken only during a pelvic examination, and sometimes without the woman's knowledge, having a mammogram is a discrete event which occurs at a time or a place other than the medical examination and thus is less likely to be confused or forgotten.

Although further studies in other populations should be done to confirm the results found here, these data provide a basis for optimism. If the findings are replicated, it appears that women's self-reports can be used accurately to monitor national changes in mammography utilization.

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Incidence of Bicycle-Related Injuries in a Defined Population

DIANE C. THOMPSON, MS, ROBERT S. THOMPSON, MD, AND FREDERICK P. RIVARA, MD, MPH

Abstract: Population-based incidence rates for head injuries and total injuries resulting from bicycle crashes were calculated in a Seattle, Washington health maintenance organization population. Overall rates were 163 per 100,00 for all injuries and 42/100,000 for head injuries. Individuals between 5 and 14 years of age are at highest risk for bicycle-related injuries. The data are presented for their potential utility in program planning. (*Am J Public Health* 1990; 80:1388–1389.)

Introduction

Bicycling is an increasingly popular sport in the United States. Injuries due to bicycling accounted for approximately 574,000 emergency room visits and 1,300 deaths in 1985, with head injuries accounting for the major morbidity and mortality.[1,2] Helmets have been shown to be an effective means of preventing these injuries.[3] Programs to promote helmet use have been successful.[4] The present study was undertaken to provide information on the incidence of these injuries for use in community bicycle helmet campaigns and safety programs.

Methods

Injured cyclists were identified during a one year surveillance of hospital emergency rooms in Seattle, Washington (December 1, 1986 to November 30, 1987). The methods are detailed elsewhere.[3] Cyclists for this study are limited to those treated at the facilities serving Group Health Cooperative of Puget Sound (GHC), a large staff-model health maintenance organization, thus allowing the calculation of population-based rates. GHC membership is demographically similar to the surrounding population in the Seattle metropolitan area, but differs from the US population by its greater educational level and underrepresentation of Blacks. Group Health Cooperative members are highly educated; 67 percent have more than a high school education. The ethnic composition of GHC membership is 91 percent Caucasian, 3 percent Black, 4 percent Asian/Pacific Islander, and 2 percent other, compared to 83.4 percent, 11.7 percent, 1.7 percent, and 3.2 percent, respectively in the US population.

GHC Central and East regions include 223,298 members who receive nearly all their outpatient, emergency, and hospital care at GHC facilities. There were two severely injured cyclists treated at the regional trauma center; these were included in the study. Review of the medical examiners' records for the study period indicated no deaths at the

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accident scene. Bicyclists injured outside the Seattle metropolitan area or treated at other area medical facilities were not included.

Exposure to cycling was measured by asking the frequency of riding and the number of miles and hours cycled per week. Self-reported mileage figures were used to calculate the mean number of miles cycled per week, and to estimate injuries per 100 miles ridden.

Incidence rates of emergency room treated injuries resulting from bicycle crashes were calculated using the mid-year 1986 population of the GHC East and Central regions.

Results

There were 364 Group Health members treated in the emergency room for bicycle-related injuries. One hundred-one (28 percent) were head injured. Thirty (29.7 percent) of the head injured experienced brain injuries; 4 percent were hospitalized. The 364 cyclists constituted 54.5 percent of all cyclists seen in the five area hospitals.

Table 1 displays age- and sex-specific injury rates. Head injury rates were highest in the 5–9 year age group while rates for all bicycle injuries were highest in the 10–14 year age group. Males had higher injury rates than females. When the incidence of bicycle-related injuries was age-adjusted to the US population, the rate was 51 per 100,000 for head injuries and 187 per 100,000 for all bicycle injuries.

Fifty-six of the 364 injured cyclists (15.4 percent) were wearing helmets at the time of the crash; only seven of the 56 head-injured were helmeted. Helmet use was very low among riders under 15 years of age (4.3 percent), but increased to 23 percent among 15–24 year olds and to 44 percent for those 25 years of age and older. There were no significant differences in rates of helmet use by sex.

Exposure to cycling is summarized in Tables 2 and 3. The injury rate per 100 miles ridden per year is highest for 5–14 year olds. Seventy-seven percent of the ER visits were between April and September.

TABLE 1—Incidence of Bicycle-Related Head Injuries and All Injuries per 100,000 population by Sex

Age (years)	Injuries (N)	Sex		Total	SE*
		Male	Female		
Head Injuries					
0–4	10	94.3	41.9	68.6	22
5–9	43	414.7	147.1	283.0	43
10–14	27	295.1	72.6	188.3	36
15–19	5	59.9	—	30.2	14
20–24	3	38.6	14.9	25.2	15
25–44	10	14.8	21.1	13.3	4
45–64	3	14.3	—	6.7	4
All ages	101			42.2	
All Bicycle Injuries					
0–4	12	121.2	41.9	82.3	24
5–9	102	907.1	414.6	671.3	66
10–14	116	1260.9	246.9	809.0	75
15–19	43	443.6	72.9	259.6	40
20–24	18	250.6	74.5	151.3	36
25–44	55	109.7	43.4	73.2	10
45–64	17	52.3	25.3	38.0	9
All Ages	364			163.0	

*Standard Error

TABLE 2—Mean Miles per Week Cycled and Injuries per 100 Miles Ridden per year According to Age and Type of Injury

Age Group (years)	Mean Miles per Week Cycled			Injuries per 100 Miles Ridden per Year
	Head Injuries	Other Injuries	All Injuries	
0–4	5.7	3.5	5.3	4.4
5–9	7.4	6.0	6.6	30.0
10–14	10.3	12.8	12.3	18.1
15–19	10.0	48.7	43.8	1.9
20–24	71.3	40.9	45.9	0.8
25–44	47.8	65.7	63.0	1.7
45–64	9.0	56.9	47.9	0.7
65+	—	—	1.0	—

TABLE 3—Distribution of Season and Week of All Bicycle Injuries

Season Injured	%	Day of Week Injured	%
January–March	13.0	Saturday/Sunday	41.0
April–June	42.0	Monday	10.2
July–September	35.3	Tuesday	11.3
October–December	9.1	Wednesday	9.1
		Thursday	16.6
		Friday	11.6

Discussion

Although helmet use has a direct effect on head injuries,[3] it does not explain why the peak ages of head injuries and overall injuries are different or why the ratio of head to all injuries is higher for young children. Our cycling exposure data, which are similar to those of other studies[1,5–7], do not explain the differences. Possible explanations are differential ascertainment of injury types and increased injury severity in older children.[8]

We assume most cycling mileage was estimated based on distance between destinations rather than actual measurement. The number of bicycle odometers used is unknown. Mileage data indicate children 5–9 years old are at highest injury risk.

Friede and co-workers (SCIPP) reported 87.8 bicycle injuries per 10,000 person years (0–19), with the injury rate highest in 6–12 year old males.[5] Our overall injury rate for this age group was 45 per 10,000. Reasons for the discrepancy are not clear. Lack of case ascertainment in the present work does not appear to be the explanation because it was a closed system of care. Other potential explanations for the differences include SCIPP'S use of all pedal cycle accidents (bicycles, tricycles, other pedal powered vehicles), sampling methods (SCIPP used a 25 percent sample of accidents over a two-year period),[5] and the proportion of all types of GHC injuries treated in the ER (40 percent) versus primary care (60 percent).[8] This latter study indicated that GHC overall rates (clinic and ER combined) appear closer to ER rates obtained by SCIPP[9] and NEOTS.[10] Two other studies[11,12] reported incidence rates of medically treated injuries similar to rates at GHC.[8] Waller (1971) reported higher bicycle injury rates. His denominator, all bicycle owners, yields higher rates than using the general population.[7]

Brain injury rates are comparable to other studies. Kraus, *et al*, reported population-based rates of brain injuries from bicycle crashes requiring hospitalization of 13.5 per 100,000 with the highest injury rates in children under age 15.[13] The rate for all brain injuries from the present work was 12.5 per 100,000.

The rates of injuries reported here are conservative since they represent only ER treated injuries. Many injured cyclists, possibly the majority, were treated at home or in outpatient clinics. Bicycling injuries contribute substantially to health care utilization and are a chief cause of recreational injury.[1,6,14–20] Children age 5–14 are at greatest risk of injury, and are an appropriate target for the application of available and effective prevention programs.[2,3,5,21]

The data presented are offered for their potential utility to the communities planning bicycle safety helmet campaigns.

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Different Views

Hypertension Prevalence and the Status of Awareness, Treatment, and Control in the Hispanic Health and Nutrition Examination Survey (HHANES), 1982–84

GREGORY PAPPAS, MD, PHD, PETER J. GERGEN, MD, MPH, AND MARGARET CARROLL, MSPH

Abstract: The prevalence rates of hypertension among adult (ages 18–74) Mexican Americans, Cuban Americans, and Puerto Ricans were estimated using data from the 1982–84 Hispanic Health and Nutrition Examination Survey (HHANES). Hypertension is defined as diastolic greater than or equal to 90 mm Hg, or systolic greater than or equal to 140 mm Hg, or currently taking antihypertensive medication. Among Mexican Americans in the Southwestern United States, 16.8 percent of the males and 14.1 percent of the females were found to be hypertensive. Among Cuban Americans in Dade County, Florida 22.8 percent of the males and 15.5 percent of the females were hypertensive. Among Puerto Ricans in the New York City area 15.6 percent of the males and 11.5 percent of the females were hypertensive. The age-adjusted rates are significantly lower than comparable rates for Whites and Blacks as measured in the second National Health and Nutrition Examination Survey (NHANES II), 1976–80. Control of hypertension in the HHANES populations fall short of the 1990 Objectives for the Nation established by the US Public Health Service 60 percent (34 percent controlled Mexican American hypertensives, 27.8 percent controlled Cuban American hypertensives, and 29 percent controlled Puerto Rican hypertensives). (*Am J Public Health* 1990; 80:1431–1436.)

Introduction

Hypertension continues to be a leading contributor to morbidity and mortality in the United States.[1] While the number of studies on the extent and nature of hypertension in the US population has increased, our knowledge of how this condition affects Hispanics has lagged behind.[2] Hispanics are the second largest minority in the United States and constitute a rapidly growing population.[3] An assessment of risk factors for hypertension has suggested that Hispanics may have high rates of the condition.[2] Surveys which have estimated the prevalence of hypertension among Hispanics have given inconclusive results.[4–8]

The drive toward better blood pressure control has been heightened by the success measured in a decline in stroke and coronary artery disease mortality.[1] The US Public Health Service has set "1990 Objectives for the Nation" including nine dealing with hypertension control. The 1990 Objectives directed towards improving health status of hypertensives state that ". . . at least sixty percent of the estimated population having definite high blood pressure (160/95) or higher should have attained successful long-term blood pressure control, i.e. a blood pressure at or below 140/90 for two or more years."[9] The extent to which Hispanics have met these goals is unknown.

This paper provides estimates of the prevalence of hypertension among three Hispanics subgroups based on the Hispanic Health and Nutrition Examination Survey (HHANES) conducted by the National Center for Health Statistics between 1982 and 1984. These estimates are compared to the published results for Whites and Blacks in the general population using the second National Health and Nutrition Examination Survey (NHANES II), 1976–80.[10] The status of awareness, treatment, and control of hypertension among Hispanics has also been estimated using HHANES data.

Methods

Survey Design

The HHANES was conducted among three Hispanic subgroups in the United States from July 1982 through December 1984. The three target populations consisted of the civilian, noninstitutionalized population of Mexican origin or ancestry in selected parts of five southwestern states (California, Arizona, New Mexico, Colorado, Texas); of Cuban origin or ancestry in Dade County (Miami), Florida; and of Puerto Rican origin or ancestry in selected counties in New York, New Jersey, and Connecticut.

The survey design of HHANES was a stratified, multistage, probability sample of persons ages six months through 74 years. Selected households within the survey were visited by a bilingual interviewer who identified eligible Hispanic families. A pre-determined sampling pattern was followed to choose specified family members to participate in the survey. A family was considered eligible if the national origin or ancestry of at least one family member met the criteria (Mexican American in the southwest, Cuban American in

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Editor's Note: See also related Different View p 1437 and editorial p 1427.

Acronyms Used:

DBP:	Diastolic blood pressure
SBP:	Systolic blood pressure
HHANES:	Hispanic Health and Nutrition Examination Survey
JNC 1984:	Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure 1984
JNC 1988:	Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure 1988
NHANES II:	Second National Health and Nutrition Examination Survey

Dade County, and Puerto Rican in the New York City area); however, the selected sample persons themselves were not necessarily of the same origin. Analyses in this paper are based on data only for those sample persons of Mexican origin or ancestry in the southwest, of Cuban origin or ancestry in Miami, Florida, and of Puerto Rican origin or ancestry in the New York City area.

Details of the HHANES sample design, selection process, operational plan, quality control procedures, and questionnaires have been previously documented.[11]

Data Collection

The HHANES included both a household interview and a physical examination conducted in a specially designed mobile examination center. As part of the household interview, sample persons were asked if they had ever been told by a physician or other health professional that they had hypertension and if they were currently using medication for treating hypertension. Upon completion of the household interview, participants were scheduled for a physical examination that included blood pressure measurement, conducted in the mobile examination center.

Systolic (first phase) and diastolic (fifth phase) blood pressure were measured to the nearest even digit using a standard mercury sphygmomanometer. Two blood pressure measurements were taken on one occasion in the mobile examination center as part of a physician's examination. Both measurements were taken with the patient seated, five minutes into the examination and five minutes apart. The average of the two readings was used for the estimates presented here.

Definitions

For the purpose of the prevalence estimates presented in this paper, persons are considered hypertensive if they meet one of the following conditions set by the 1988 Report of the Joint National Commission on Detection, Evaluation, and Treatment of High Blood Pressure (JNC 1988): 1) a diastolic blood pressure of greater than or equal to 90 mm Hg; 2) a systolic blood pressure greater than or equal to 140 mm Hg; 3) reporting currently using anti-hypertensive medication (regardless of blood pressure measurements).[1] The 1990 Objectives use a second definition for hypertension setting the thresholds at diastolic greater than or equal to 95 mm Hg and systolic greater than or equal to 160 mm Hg. This second definition for hypertension (160/95) is used to assess the levels of awareness, treatment, and control of hypertension.

Prevalence estimates for the specific categories of adult blood pressure levels follow the JNC 1988 guidelines. These categories are listed and defined in the Appendix. Blood pressure levels are reported as a percent of the HHANES target population and as a percent of the hypertensives in those populations.

Hypertension prevalence estimates are available for Whites and Blacks in the general population based on the NHANES II (1976–80). These estimates have been previously published in a report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, 1984 (JNC 1984).[10] The JNC 1984 estimates for hypertension were based on the average of three pressures (taken with the examinee sitting, lying, and again sitting). A caveat for the comparisons is that the JNC 1984 estimates included a small number of Hispanics. However, the removal of Hispanics from the sample does not produce estimates that show statistically significant differences from the estimates published in JNC 1984 report.

The percent of hypertensives under control on medication is defined as: the number of hypertensives who report taking medication and whose blood pressure is less than the threshold of 140/90 mm Hg (numerator) divided by the total of the hypertensives (denominator). Control is reported as a proportion of both definitions of hypertension, using the 140/90 and 160/95 thresholds.

The higher threshold for hypertension determines the denominator for the status variables (awareness, treatment, control). A person is considered aware of their hypertension if they report having ever been told by a physician or other health professional that they have high blood pressure or hypertension. Those who report they are currently taking antihypertensive medication are considered treated.

Response Rates

Among those selected for HHANES and eligible (of Hispanic origin), ages 18 to 74 years, there were 5,130 Mexican Americans, 1,560 Cuban Americans, and 1,942 Puerto Ricans. Of the selected persons, the proportion of persons interviewed was 77.4 percent, 72.6 percent, and 77.9 percent for Mexican Americans, Cuban Americans, and Puerto Ricans, respectively. Two blood pressure measurements were obtained on 82.3 percent, 75 percent, and 77.7 percent of the interviewed persons in the Mexican American, Cuban American, and Puerto Rican samples, respectively. No significant differences were noted with regard to age, sex, and antihypertensive medication status between persons interviewed and persons with two blood pressure measurements in any of these subgroups.

Systematic Bias in BP Measurements

The tendency toward blood pressure readings ending in zero was identified as a problem in HHANES (diastolic 23.8 percent, systolic 27.6 percent). Further analysis of this systematic bias was made around the critical values used to define hypertension: 90 mm Hg diastolic and 140 mm Hg systolic. Diastolic 86 mm Hg and 88 mm Hg were read more often than either 90 mm Hg or 92 mm Hg. Systolic 140 was read more often than 138. Thus it appears that the examiners tended to read below the level defining hypertension on the diastolic reading and above the level defining systolic hypertension. Therefore, the previously noted systematic bias may be thought to have an equivocal effect on the point estimates presented here.

Statistical Analysis

Sampling weights were used when calculating point estimates in order to account for individual selection probabilities and adjustments for non-response, non-coverage, and poststratification which resulted from the complex survey design used in HHANES. When the appropriate weights are used, the samples are representative of the Mexican origin population in the five southwestern states, the Cuban origin population in Dade County, Florida, and the Puerto Rican origin population in the New York City area.

The complex survey design used in the HHANES tended to increase the variance which would have been obtained through simple random sampling.[12] The complex survey design was accounted for by using an average design effect to adjust the variance calculated under the assumption of simple random sampling. As described by Kovar,[13] the design effect is the ratio of the variance of a statistic from a complex sample to the variance of the same statistic from a simple random sample. The value of the design effect is an indicator of the impact of the complex sample design on the variance.

The average design effect for hypertension was 1.0 for all three Hispanic subgroups. The complex design, therefore, has little effect on the variances and one could consider assuming simple random sampling for these analyses. A t-test was used to compare point estimates. All data analyses were done using programs accessible through SAS.[14]

The direct method was used for age adjustment. The reference population was the civilian noninstitutionalized population of the United States at the midpoint of the NHANES II, March 1, 1978.

Results

Prevalence of hypertension from HHANES is presented by subgroup, sex, and age in Table 1. These estimates represent approximately 775,000 hypertensive Mexican Americans in the southwest, 56,400 hypertensive Cubans in Dade County, Florida, and 78,000 hypertensive Puerto Ricans in the New York City area at the 1983 population levels.

Expected age and sex differences in hypertension were observed for the three Hispanic subgroups. The prevalence of hypertension was higher in older age groups for each of the three Hispanic subgroups. The rates of hypertension were age-adjusted, using the US population in 1978 as a reference (the mid-point of NHANES II). Men had significantly higher age-adjusted prevalence estimates for hypertension compared to women in the Mexican American (95% CI = 0.2, 6.2) and Cuban American (95% CI = 1.4, 12) subgroups. Age-specific comparisons between sexes suggest the same conclusion.

Table 2 presents the distribution of blood pressure levels regardless of medication status, among men and women in the Hispanic subgroups. The results are similar for all three Hispanic subgroups. The great majority of individuals in the HHANES populations have blood pressure measurements in the normal range. Mild diastolic elevation (DBP 90-104) ranges from 2.7 percent to 11.1 percent in the HHANES populations. Isolated systolic hypertension (SBP > = 160) was discovered in a very small portion of the population (less than 1.2 percent).

The distribution of blood pressure levels among hypertensives is presented in Table 3. By far the largest numbers of hypertensives are found in the mild hypertensive category (DBP 90-104), approximately 40 percent. Taken together, those with borderline isolated systolic hypertension (SBP 140-159) and isolated systolic hypertension levels (SBP > = 160) made up over one-third of all the Hispanic hypertensives. Less than 20 percent of all hypertensives (140/90) were controlled by medication.

A number of statistically significant results were observed comparing hypertension rates from HHANES with Whites and Blacks in the general population as measured in NHANES II (1976–80). Hypertension is more prevalent among Whites or Blacks than it is among Mexican Americans, Cuban Americans, or Puerto Ricans (see Table 4). Individual comparisons using the age-adjusted estimates revealed that each of the three Hispanic subgroups had statistically significant lower levels of hypertension than did either Blacks or Whites. Comparisons of age-specific rates suggest the same conclusion.

TABLE 1—Hypertension* Prevalence by Hispanic Subgroup, Sex and Age

	Males			Females		
Sex, Hispanic Origin, and Age (years)	Number of examined persons	Percent with hypertension	Standard error of the percent	Number of examined persons	Percent with hypertension	Standard error of the percent
Mexican Americans						
18–74	1553	16.8	1.2	1965	14.1	0.9
18–74 age adjusted		22.9	1.1		19.7	0.9
18–24	342	5.1	1.6	417	0.5	0.4
25–34	423	10.4	2	527	4.9	1.1
35–44	246	12.3	2.8	333	7.6	1.7
45–54	268	28.5	2.8	350	24.1	2.2
55–64	191	47.2	3.6	219	44.9	3.4
65–74	83	59.6	6.4	119	66.8	4.6
Cuban Americans						
18–74	398	22.8	1.2	498	15.5	0.9
18–74 age adjusted		20.5	2		13.8	1.5
18–24	55	1.1	0.9	54	0	0
25–34	62	5.5	2	73	1.1	0.8
35–44	52	13.7	3.3	94	5.2	1.5
45–54	114	37.5	2.4	116	15.2	1.8
55–64	74	41.4	3.1	97	35.6	2.6
65–74	41	46.3	4.2	64	49.8	3.3
Puerto Ricans						
18–74	495	15.6	1.1	835	11.5	0.7
18–74 age-adjusted		19.7	1.8		18	1.3
18–24	112	1.1	0.7	188	0.6	0.4
25–34	107	4.6	1.6	171	1.9	0.8
35–44	73	17.4	3.6	152	7.1	1.6
45–54	102	26.4	2.5	174	23.7	1.8
55–64	78	51.1	3.2	97	47	2.7
65–74	23	**	**	53	55.2	3.8

SOURCE: NCHS, HHANES, 1982–84

*Defined as the average of two blood pressure measurements >= 140/90 mm Hg or currently taking antihypertensive medication.

**Sample size too small to produce reliable estimates.

TABLE 2—Distribution of Blood Pressure Level Regardless of Medication Status

	Mexican American		Cuban American		Puerto Rican	
Blood Pressure Level	Males n=1553 % SE	Females n=1965 % SE	Males n=398 % SE	Females n=498 % SE	Males n=495 % SE	Females n=835 % SE
Normal Blood Pressure*	72.7 ± 1.4	85 ± 0.9	67.8 ± 1.4	81.4 ± 1	78.7 ± 1.3	88.2 ± 0.8
High Normal Blood Pressure*	12.3 ± 1	4.9 ± 0.5	13 ± 1	6.6 ± 0.6	6.9 ± 0.7	3.7 ± 0.4
Mild High Blood Pressure	8.8 ± 0.9	4.1 ± 0.5	11.1 ± 0.9	4.8 ± 0.6	10.2 ± 0.9	2.7 ± 0.4
Moderate High Blood Pressure	1.0 ± 0.3	0.2 ± 0.1	1.7 ± 0.4	0.7 ± 0.2	0.3 ± 0.2	0.4 ± 0.1
Severe High Blood Pressure	0.2 ± 0.1	0.1 ± 0.1	0.7 ± 0.2	0 ± 0	0.1 ± 0.1	0 ± 0
Borderline Isolated Systolic Hypertension	4.7 ± 0.7	5 ± 0.5	4.3 ± 0.6	5.3 ± 0.6	2.8 ± 0.5	4.2 ± 0.5
Isolated Systolic Hypertension	0.4 ± 0.2	0.6 ± 0.2	1.2 ± 0.3	1.2 ± 0.3	0.9 ± 0.3	0.6 ± 0.3

*Includes those hypertensives who are controlling their blood pressures.
SOURCE: NCHS, HHANES, 1982–84

Results of comparisons of the distribution of blood pressure levels (regardless of medication status) indicate that Whites or Blacks in NHANES II generally had higher levels in each elevated blood pressure category when compared to Hispanics. Whites and Blacks (both sexes) were two or three times more likely to have mildly elevated diastolic blood pressure (DBP 90-104) than each of the Hispanic subgroups. Among White men and women, as found in the JNC 1984 report, 18.4 percent and 10.8 percent, respectively had mildly elevated diastolic blood pressure. Mildly elevated diastolic blood pressure has been found in 20.6 percent and 17.9 percent of Black men and women, respectively. Whites and Blacks were also more likely to have moderately (DBP 105-114) and severely elevated (DBP > = 115) blood pressures, compared to Hispanics.

The proportion of hypertensives (160/95) aware, treated, and controlled is presented by sex and Hispanic subgroup in Table 5. Awareness was high among women, over 95 percent in each of the three groups. Within each Hispanic subgroup, women hypertensives reported having been told about their hypertension more often than men. A substantial portion of Mexican American and Puerto Rican male hypertensives, i.e. 32.1 percent and 21.3 percent, respectively, are unaware of their condition.

Similarly, a larger number of Mexican American and Puerto Rican men were on no medication for their condition, compared to women in those subgroups. Cuban American hypertensive men were more likely to be on medication than hypertensive men in the other two Hispanic subgroups.

Control rates among hypertensives using the 1990 Objectives definition were generally low. With the hypertension threshold at 160/95 and control threshold at 140/90, the proportions of male hypertensives under control were 22.6 percent, 26.1 percent, and 11.9 percent for Mexican Americans, Cuban Americans, and Puerto Ricans, respectively (see Table 5). Higher rates of control were found among hypertensive women as compared to men, except in the Cuban subgroup where the level of control was similar for male and female hypertensives. No group attained the 1990 Objectives of 60 percent controlled.

TABLE 3—Percent of Individual Blood Pressure Readings (systolic and diastolic) around Critical Values for Definition of Hypertension

Diastolic Blood Pressure	Percent	Systolic Blood Pressure	Percent
86	30.8	136	24.6
88	31.7	138	15.4
90	17.5	140	25.4
92	10.8	142	21.4
94	9.1	144	13.3

SOURCE: NCHS, HHANES 1982–84

Using a less stringent threshold for control (160/95), men still fall short of the objective of 60 percent control; 38.4 percent, 50.6 percent, and 19.7 percent of the hypertensive males (160/95) were controlled for Mexican Americans, Cuban Americans, and Puerto Ricans, respectively.

Discussion

The major finding of this study is that the HHANES populations had significantly lower proportions of hypertensives than did Whites or Blacks in NHANES II. This finding is supported by studies showing lower cardiovascular mortality rates among Mexican Americans and Puerto Ricans.[4,15] However, cardiovascular diseases remain a leading cause of morbidity and mortality in these groups and the importance of hypertension should not be underestimated.[16]

Lower rates of hypertension are contrary to the expectations suggested by levels of risk factors for the condition among Hispanics. Considering the generally lower socioeconomic status of Hispanics,[2] the high prevalence of obesity,[17] and the high prevalence of diabetes,[18] we would anticipate hypertension rates to be higher than Whites. A study of risk factors for hypertension using HHANES data may shed light on this problem.

TABLE 4—Hypertension Prevalence Estimates NHANES II and HHANES (ages 18–74 years)

	Male		Females	
	%	SE	%	SE
*NHANES II**				
White	32.6	1.4	25.3	0.9
Black	37.9	2.2	38.6	1.8
*HHANES***				
Mexican American	22.9	1.1	19.7	0.9
Cuban American	20.5	2	13.8	1.5
Puerto Rican	19.7	1.8	18.0	1.3

SOURCE: NHANES II, 1976–80 (JNC 1984) NCHS, HHANES, 1982–84
*JNC 1984
**Age-adjusted to 1978 population

TABLE 5—Hypertension* Awareness, Treatment, and Control** Status among Three Hispanics Subgroups

	Males		Female		Female/ male % difference	
	%	SE	%	SE		(95% CI)
Mexican American						
Awareness	67.9	5.0	95.0	1.8	27.1	(15.8, 38.4)
Treated	49.3	5.3	86.0	2.9	36.7	(23.9, 49.5)
Controlled	22.6	4.4	43.6	4.1	21.0	(8.3, 33.7)
Cuban Americans						
Awareness	87.2	2.6	95.1	1.6	7.9	(1.4, 14.4)
Treated	70.1	3.6	79.1	3.0	9.0	(−0.9, 18.9)
Controlled	26.1	3.5	29.5	3.3	3.4	(−6.8, 13.6)
Puerto Ricans						
Awareness	78.7	3.8	96.1	1.2	17.4	(9, 25.8)
Treated	41.0	4.6	85.5	2.2	44.5	(33.7, 55.3)
Controlled	11.9	3.0	41.6	3.1	29.7	(20.6, 38.8)

SOURCE: NCHS, HHANES, 1982–84

*Hypertension is defined as either >=160/95 or reported currently taking antihypertensive medication.

**Control is defined as a threshold of 140/90.

Previous surveys comparing blood pressure levels of Hispanics with Whites have given inconclusive results. The results of the San Antonio Heart Study, conducted between 1979–82, suggests that Mexican American males had a prevalence of hypertension approximately equal to Whites, and Mexican American females had lower rates than White females.[4] In Orange County, California, Hispanics and non-Hispanics were found to have similar systolic and diastolic blood pressure.[5] The California Hypertension Survey (1978) found that Hispanic men had slightly lower rates of hypertension than White men. Among women, the prevalence of hypertension was higher in Hispanic women than in White women in the younger age groups and somewhat lower in older age groups.[6] The one study of blood pressure among Puerto Ricans was small in scale and nonrepresentative.[7] No study of Cuban American blood pressure has previously been published. The conclusions of these earlier studies which differ from those presented here may be due to the larger and more representative sample selected in HHANES.

The limitations of these data include the facts that: 1) hypertensives controlling their blood pressure without medication are not included in the definition; 2) only the civilian, non-institutionalized population ages 18–74 are included; 3) the sampling universe defined in HHANES does not include all persons of Hispanic origin; and 4) measures were taken on only one occasion. The use of blood pressure measurements on one occasion limits both the definition of hypertension and the "1990's" definition of control. Clinical definitions require more than one elevated measurement and the "1990 Objectives" define long-term control as "at or below 140/90 for two or more years."

The comparison of the prevalence of hypertension among Whites, Blacks, and Hispanics presented here must address the possibility of temporal changes. The NHANES II and HHANES were conducted five years apart. The prevalence of hypertension in the general population has remained relatively stable over the past 20 years.[19] The incidence of hypertension also seems to have been stable for this period as measured in the Framingham study population.[20] Comparisons between the temporally separated NHANES II and HHANES are useful if we reasonably assume the rates among Hispanics have also been stable.

No comparisons of Whites or Blacks with the three Hispanic subgroups were attempted for awareness, treatment, and control status. Direct comparison of HHANES and NHANES II for the status of awareness, treatment, and control of hypertension is inappropriate because of well described secular trends. Over the past decade, awareness of hypertension has increased, more people have begun treatment, and control has improved.[21–23] These important changes were happening during the interval between NHANES II (1976–80) and HHANES (1982–84). To the extent that US Hispanics have participated in the transition, we would expect that awareness, treatment, and control of hypertension also would have improved since the time when NHANES II was conducted.

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APPENDIX

Diastolic Blood Pressure (DBP) (mm Hg)	Category*
<85	Normal Blood Pressure
85–89	High Normal Blood Pressure
90–104	Mild Hypertension
105–114	Moderate Hypertension
≥115	Severe Hypertension
Systolic Blood Pressure (SBP) (mm Hg) when DBP <90 mm Hg	Category*
<140	Normal Blood Pressure
140–159	Borderline Isolated Systolic Hypertension
≥160	Isolated Systolic Hypertension

*A classification of borderline isolated systolic hypertension (SBP 140 to 159 mm Hg) or isolated systolic hypertension (SBP ≥ 160 mm Hg) takes precedence over a classification of high normal blood pressure (DBP 85 to 89 mm Hg) when both occur in the same individuals. A classification of high normal blood pressure (DBP 85 to 89 mm Hg) takes precedence over a classification of normal blood pressure (SBP <140 mm Hg) when both occur in the same individual.

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IOM Conference on Health Status Assessment: Call for Papers

The Institute of Medicine of the National Academy of Sciences has announced a call for papers for its September 1991 conference on Advances in Health Status Assessment. Building on the outcome of two previous conferences (1986 and 1988) this conference will concentrate on the use and usefulness of health status and health-related quality of life measures in clinical practice, especially ambulatory care, and in clinical research that will directly facilitate the application of these measures by practitioners. The following issues are of particular interest, although others will be considered, and empirical research papers are especially invited.

- The use of measures of health status and health-related quality of life as aids in screening, planning patient care, monitoring individual patient outcomes of care, or evaluating quality of care in ambulatory clinical settings;
- Improvements in the definition, reliability, validity, and other methodologic or statistical aspects of health status and outcome measures, as they related to clinical settings.

The deadline for submission of abstracts of at least 2500 words is February 15. Author notification will be on or about April 1, with first drafts due June 1 and final publishable versions due August 1. For further information, contact Kathleen N. Lohr, PhD, or Jo Harris-Wehling, MPA, IOM, NAS, 2101 Constitution Avenue, NW, Washington, DC 20418; (202) 334-2165.

A Note on the Measurement of Hypertension in HHANES

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Abstract: Using data from the HHANES, we found the rates of elevated blood pressure readings on clinical examination to be extremely low for a sample of Mexican American and Puerto Rican women. The prevalence rates were one-fourth to one-fifth the rates found for a comparable sample of White women from NHANESII. These findings are discrepant with the little that is known about hypertension prevalence among Hispanics and with estimates of hypertension prevalence for Mexican Americans and Puerto Ricans drawn from NHANESII. While our HHANES samples women had much lower rates of clinical high blood pressure than Whites, they reported hypertension histories in excess of Whites. Rates of medicine usage among Hispanics were insufficiently large for effective treatment to explain the disparity. The prevalence estimates increased, but the relative discrepancies remained when we altered our sample specifications and clinical high blood pressure measure. A possible explanation for these discrepancies is that few physicians performed the majority of blood pressure readings in our HHANES sample. This may have been statistically inefficient. The discrepancies noted suggest that HHANES may not be a reliable source of information on hypertension among Hispanic women. (*Am J Public Health* 1990; 80:1437–1442.)

Introduction

Hypertension is one of the leading causes of death in the United States. It is an illness that can be easily diagnosed and in the aggregate has responded well to medication. Over the past 20 years, there has been an increase in the proportion of the hypertensive population diagnosed as having hypertension, an increase in the proportion using antihypertensive medicine, and a decrease in the incidence of clinical hypertension.[1] Despite the improvements in the diagnosis, treatment, and control of hypertension in the United States, it remains a persistent health problem, especially for Blacks.[1-3]

Less can be said about the historical or current trends in hypertension for the various Hispanic populations. Most available data have been local area, small-sample studies[4-8] which cannot be generalized to the national population. While Hispanic respondents have often been identified in national health surveys, their numbers were usually too small to analyze well. Often, the definition of the Hispanic population is an aggregation of all Spanish-speaking groups, a practice which ignores the real differences among the various Hispanic groups.[9-11]

With the release of data from the 1982-84 Hispanic Health and Nutrition Examination Survey (HHANES), the potential to apply sophisticated analytical approaches to understanding disease patterns within Hispanic populations is enhanced. HHANES is the only survey that has both large representative samples of members of Hispanic groups within the United States and includes objective measures of health through the examination data, rather than relying only on self-reports. Thus, the validity of the clinical data is critical for the research and policy questions HHANES is intended to address. However, as this paper suggests, there may be problems with the HHANES clinical blood pressure data for women.

Methods

The focus of this analysis is the hypertension data in HHANES. However, to provide a comparison population, information from the National Health and Nutrition Examination Survey, 1976–80 (NHANESII) is also used. Both the NHANESII and HHANES data sets utilized complex, multistage, stratified, clustered samples of defined populations. HHANES differs from NHANESII and previous health interview surveys conducted by the National Center for Health Statistics in that it was not a national probability survey, but a survey of three special subgroups of the population who reside in three geographically distinct regions: Mexican Americans in the southwest United States, Puerto Ricans in the New York area, and Cuban Americans in Miami, Florida. Like NHANESII, the data include an interview and a direct medical examination for a sample of civilian, noninstitutionalized persons 6 months to 74 years of age. (For more details on the sampling procedures used in HHANES and NHANESII, see the NCHS plans for operation.[12,13])

Our ongoing work on the health status of women in their childbearing years led us to the discovery of potential problems with the HHANES blood pressure data. Our sample included all currently menstruating women, who were not pregnant at the time of the survey, and were between the ages of 12 and 54 years. The White and Black members of our sample were drawn from NHANESII while the Puerto Rican and Mexican American sample members were drawn from HHANES. Only respondents who identified themselves as Mexican in the southwest sample or Puerto Rican in the northeast sample were used in the analysis. The resulting sample sizes were 1,700 Mexican Americans, 757 Puerto Ricans, 2,963 Whites, and 509 Blacks. Persons of Hispanic origin have been excluded from the White and Black populations. Due to small numbers, Cuban American women were excluded.

For the HHANES examinations, blood pressure measurements were obtained using procedures recommended by the American Heart Association. For the Black and White populations (NHANESII), three readings were taken (sitting, recumbent, sitting), while for the Mexican American and Puerto Rican populations (HHANES), two readings were taken (both sitting). Additional questions on hypertension history, treatment history and compliance, and current medicine usage were ascertained through the detailed medical history interview. Averages of the available blood pressure

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readings and some of the interview information provide the basis for our determination of hypertension status. The following indicators of hypertension are used in the analysis:

- Respondent has clinical high blood pressure (HBP). In our initial sample clinical HBP was defined as mean arterial pressure (MAP) greater than 105 where MAP = [systolic + (2 × diastolic)]/3.[14,15] As explained below in the text, in later specifications we defined it as systolic ≥ 140 of diastolic ≥ 90.
- Respondent is currently taking antihypertensive medicine.
- Respondent has ever been told by a doctor that she has high blood pressure or hypertension.

It is standard procedure to determine hypertension status on the basis of the first two of these three indicators.[1] Thus, we initially combined them to identify hypertensives. However, when the clinical data appeared questionable, we also looked at the third indicator to learn if the history and clinical data were consistent.

Standard errors are calculated under the assumption that the NHANESII data are simple random samples of the population. The complex sample design implies that these standard errors are almost certainly too small, by a factor that could be as large as $\sqrt{2}$. Initially we attempted to account for the complex samples design using both Taylor series expansion and balanced half sample methods.[16-20] Estimated design effects for the Black, Mexican American, and Puerto Rican samples were often implausibly low (less than 1). These results are consistent with those found by NCHS staff.[21,22] Following NCHS suggested guidelines,[23] which involve averaging design effects across subsamples, did not yield more reasonable results. We thus thought it most reasonable to simply report standard errors without taking into account the complex sample design while warning the reader to interpret results cautiously.

Using the direct method,[24] summary statistics for Blacks, Mexican Americans, and Puerto Ricans are standardized to the age distribution of our White sample.

Results

HHANES Clinical High Blood Pressure Data

Tables 1 and 2 provide selected measures of hypertension according to race and Hispanic ethnicity. Not only do Mexican Americans and Puerto Ricans have the lowest prevalence of elevated high blood pressure readings (clinical HBP), but the overall rate for Mexican American women is only one-third the White rate (1.7 versus 5.1) while the Puerto Rican rate is not even one-fourth of the White rate (1.2 versus 5.1). Even allowing for large design effects, these differences are clearly significant. The rates of clinical HBP also look questionable when they are examined by age (Table 2). Among Puerto Rican women, there are no cases of high blood pressure under the age of 35. Among Mexican American women there are hypertensives in all but the youngest group (<15) but, unlike Black or White women, even at the oldest ages hypertension prevalence does not approach double digits.

TABLE 2—Prevalence of Clinical High Blood Pressure by Race/Ethnicity and Age

	Hispanic		Non-Hispanic	
Age (years)	Mexican American	Puerto Rican	White	Black
<15	0.0	0.0	0.7	0.0
15–24	0.1	0.0	1.1	1.7
25–34	2.0	0.0	3.1	7.3
35–44	3.3	3.1	9.7	22.8
45–54	5.0	9.8	21.9	48.4
Total[a]	1.7	1.2	5.1	9.2

[a] Standardized to the White non-Hispanic population
SOURCE: HHANES, 1982–1984 and NHANESII, 1976–1980.

The low magnitudes and unusual age patterns of hypertension prevalence we observed in HHANES based on clinical high blood pressure were unanticipated. That both Hispanic populations had prevalence rates that were not simply lower than, but were actually only a fraction of, the White was unexpected. This led us to question whether Mexican American and Puerto Rican women do indeed have extremely low rates of hypertension or whether the clinical high blood pressure data for Puerto Rican and Mexican American women in HHANES were downwardly biased.

To examine the matter further we conducted the following exercises: 1) we compared our findings to those in the literature on high blood pressure among Mexican Americans and Puerto Ricans; 2) we compared the clinical high blood pressure data in HHANES to that reported for the Mexican American and Puerto Rican respondents in NHANESII; 3) we considered other measures of hypertension in HHANES; 4) we checked the sensitivity of our prevalence estimates to our sample specifications and clinical measure of high blood pressure; and 5) we considered the possibility that study effects, specifically physician effects, influenced the HHANES clinical data.

Comparisons with Other Studies

A few recent studies have examined hypertension in the Mexican American and Puerto Rican populations.[4-8] The one Puerto Rican study was of a highly selected sample in the Bronx (participants at a health fair), from which it is difficult to generalize. However, hypertension rates were very high, with the majority of sample members hypertensive. Most studies of Mexican Americans have been local area studies and, thus, cannot be generalized to the US Mexican population. These local area studies have not usually focused

TABLE 1—Selected Measures of Hypertension by Hispanic Ethnicity and Race, Age Standardized to the White Non-Hispanic Population, with Standard Errors

	Clinical HBP				Current Medicine Usage				HBP History				Clinical HBP + Medicine Usage			
	Mx	PR	Wh	Bl	Mx	PR	Wh	Bl	Mx	PR	Wh	Bl	Mx	PR	Wh	Bl
Percent Hypertensive (Standard Error)	1.7 (.3)	1.2 (.4)	5.1 (.4)	9.2 (1.3)	2.2 (.4)	2.1 (.5)	2.7 (.3)	5.2 (1.0)	3.7 (.8)	17.2 (1.4)	12.8 (.6)	19.3 (1.7)	3.5 (.4)	2.8 (.6)	6.5 (.4)	12.2 (1.4)

SOURCE: Menstruating, non-pregnant women ages 12-54 HHANES, 1982-84 and NHANESII, 1976-80 specifically on women and, when women have been included, the age stratification is not as detailed as in our study. Nevertheless, data from the local area studies indicate that the Mexican Americans do not have extremely low rates of hypertension. For example, both a Laredo, Texas[4] and a Sacramento, California[7] study found rates of hypertension for Mexican Americans to be intermediate between Whites and Blacks, while a study comparing Mexican Americans and non-Hispanic Whites in San Antonio, Texas found them to have roughly comparable rates.[6] Point estimates were slightly lower for Mexican American women compared to non-Hispanic Whites, but this difference was not statistically significant. We were able to simulate a portion of the Laredo, Texas study using the HHANES data. Using the same sample specifications and the same measure of clinical hypertension as used in that study (diastolic ≥ 95), we were able to calculate a prevalence estimate for one age group, women 40–49 years. While in the Laredo study the point estimate for this group was 7.9 percent, using HHANES data, ours was only 1.9 percent.

Hispanics in HHANES versus NHANESII

Using the NHANESII data, Table 3 presents several measures of hypertension for Mexican American, Puerto Rican, Black, and White women. It is important to note sample sizes. There are only 35 Puerto Rican women in NHANESII, too few for analysis of a rare event such as hypertension. There are 140 Mexican American women which is much less than what one could find in HHANES, but enough for rough comparisons.

Unlike in HHANES, Hispanic hypertension rates in NHANESII are fairly comparable to those found among White women. The point estimates for Hispanic women in NHANESII are actually higher than those for Whites. Using NHANESII, the ordering of prevalence rates by racial/Hispanic ethnic identification is what one might expect based on the local area studies and what is more generally known about the relative health and socioeconomic status of US Black, Whites, Puerto Ricans and Mexican Americans.[25]

Other Measures of Hypertension in HHANES

HHANES has several indicators of hypertension besides the clinical measure, most prominently self-reported history data. The clinical data and the hypertension history data in HHANES appear discrepant (see Table 1). If Mexican American women had a low prevalence of hypertension, we might expect to see a low prevalence of positive hypertension histories. Mexican American and White women report positive hypertension histories in roughly equal proportions, while Puerto Rican women report a positive hypertension history significantly *more* often. Inspecting the data within age groups, we observed additional anomalies. Examples are (not reported in the tables): among the 27 Puerto Rican women 48 to 50 years of age, 13 (43 percent) report a history of high blood pressure; yet only two currently have an elevated clinical reading and three are taking antihypertensive medicine. Among the 42 Mexican American women in this age interval, 15 (33 percent) report a history of high blood pressure; yet only nine are defined as currently hypertensive (four with clinical high blood pressure and five currently using antihypertensive medication). Some of the "history only" women were probably reporting an experience with a finite hypertensive period such as pregnancy-induced hypertension. However, to explain the entire discrepancy by this fact, one would have to assume that Mexican American and Puerto Rican women are far more prone to such periods than are Black or White women, since discrepancies of this magnitude were not found among Blacks or Whites. Similarly, to infer recall bias explains the entire difference would imply that Hispanics are more likely to misreport than Blacks or Whites, and that both Hispanic groups misreport in the same direction. This seems unlikely and is also unsupported by the Hispanic data in NHANESII. Among Mexican Americans and Puerto Ricans in NHANESII, there is a −0.5 point and a 3.5 point difference, respectively, between the percent reporting a history of hypertension and those with evidence of current hypertension. The differences in the HHANES data are much greater at 10.2 points (Mexican Americans) and 14.4 points (Puerto Ricans).

We also considered the effects relevant secular trends might have on our findings. The NHANESII and HHANES surveys were implemented several years apart. During this time efforts were made to educate the public about the effects of hypertension and the importance of treatment. The secular trends in awareness would lead us to expect to find higher percentages reporting a history of hypertension and higher percentages reporting experience with antihypertensive treatment in HHANES compared to NHANESII, but hypertension prevalence estimated as the combination of elevated readings and medicine usage would not be expected to be lowered dramatically. Comparing the HHANES and NHANESII results, the proportions of self-reported histories did increase between the two surveys, as expected, but hypertension prevalence was lowered far beyond what one might expect. The rate of elevated clinical readings decreased 75 percent for Mexican Americans and 86 percent for Puerto Ricans; the prevalence rate (elevated readings plus medicine usage) decreased 54 percent for Mexican Americans and 68 percent for Puerto Ricans.

Questions are also raised by comparisons of the medicine usage and hypertension prevalence data. If the clinical data are taken at face value, the figures in Table 1 show that 63 percent of hypertensive Mexican American women and 75 percent of hypertensive Puerto Rican women take medicine. The corresponding figure for White women is 42 percent and for Black is 43 percent. These figures suggest that hypertensive Mexican American and Puerto Rican women (women who are members of populations usually described as medically underserved) undergo medical treatment in higher

TABLE 3—Selected Measures of Hypertension for Mexican American and Puerto Rican Women: Data from NHANESII

	Sample Size				Clinical HBP		HBP History		Clinical HBP + Medicine Usage			
	Mx	PR	Wh	Bl	Mx	PR	Mx	PR	Mx	PR	Wh	Bl
Total[a] (Standard errors)	140	35	2693	509	6.9 (2.1)	8.7 (4.8)	7.1 (2.2)	12.2 (5.5)	7.6 (2.2)	8.7 (4.8)	6.5 (.4)	12.2 (1.4)

[a] Standardized to the White non-Hispanic population.

SOURCE: NHANESII, 1976–80.